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Original Research

Effect of 4.7-mg Deslorelin Acetate Implant on Blood Lipids and Steroid Hormones in Cockatiels (*Nymphicus hollandicus*)

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Abstract: Deslorelin acetate is a gonadotropin-releasing hormone agonist commonly used for reproductive suppression in birds. Female psittacine birds routinely undergo vitellogenesis, with resulting high blood lipids, which may predispose them to several lipid disorders, including atherosclerosis. This study evaluated the effects of deslorelin implants on blood lipids and steroid hormones in cockatiels (*Nymphicus hollandicus*). Sixteen female cockatiels were randomly assigned to receive a 4.7-mg deslorelin implant subcutaneously or a sham procedure. Plasma samples were collected at baseline and at 1, 2, 4, and 6 months postimplantation. Lipid and lipoprotein profiling were performed by high-resolution polyacrylamide gel electrophoresis, and steroid hormones were analyzed via liquid chromatography tandem spectrometry. Deslorelin significantly reduced plasma triglycerides, total cholesterol, intermediate-density lipoproteins, and high-density lipoproteins at 1 month postimplantation compared with controls, with some effects persisting up to 2 months for intermediate-density lipoproteins. Four of 8 cockatiels were found to be hyperlipidemic in the deslorelin group over the 6-month treatment period compared with 8 of 8 cockatiels in the control group. Progesterone concentrations were lower in the deslorelin group. Estradiol concentrations decreased transiently at 1 month but did not reach statistical significance. These findings highlight deslorelin's potential to modulate lipid metabolism in female cockatiels and suggest a temporal effect lasting 1–2 months. This study provides novel insights into the metabolic effects of deslorelin beyond its reproductive role and may have implications for managing hyperlipidemia in avian species.

Key words: vitellogenesis, cholesterol, triglycerides, deslorelin, avian, psittacine, cockatiel, *Nymphicus hollandicus*

INTRODUCTION

Reproduction-associated lipid accumulation disorders are commonly reported in female psittacine birds, although published data documenting overall prevalence remain limited. In reproductively active females, estrogen stimulates an increase in plasma concentrations of total calcium, proteins, cholesterol, phospholipids, and triglycerides, while also increasing hepatic production of vitellogenin and very low-density lipoprotein (VLDL).[1,2] Female psittacine birds have a higher prevalence of hyperlipidemia associated with frequent and dysregulated vitellogenesis.[3] This prolonged hyperlipidemia may increase the risk of developing lipid accumulation disorders, such as atherosclerosis and hepatic lipidosis.[4]

During vitellogenesis, the liver synthesizes yolk precursor proteins and lipids, which are then packaged into specific triglyceride-rich lipoproteins, the yolk-targeted VLDLs (VLDLys), and phospholipid-rich and protein-rich lipoproteins, the vitellogenins, for transport via the bloodstream to developing oocytes. Under the influence of steroid hormones, their circulating concentrations increase markedly at the onset of egg laying and decline during laying pauses.[5] These lipoproteins are resistant to lipoprotein lipase in the blood[2,6] and thus can lead to more persistent elevations in blood lipids when the

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process is dysregulated or maladaptive, as is suspected to occur in psittacine birds under human care.[3]

Administration of an exogenous gonadotropin-releasing hormone (GnRH) superagonist suppresses the hypothalamic-pituitary axis, thereby downregulating reproductive activity. As a result, concentrations of vitellogenic compounds such as VLDLys, vitellogenins, triglycerides, phospholipids, proteins, and other associated metabolites are expected to decrease. Deslorelin acetate implants are one of the most commonly used GnRH superagonists to suppress the hypothalamic-pituitary-gonadal axis. Deslorelin initially triggers an overstimulation of pituitary GnRH receptors, causing a temporary surge in luteinizing hormone (LH) and follicle-stimulating hormone production. These gonadotropins then stimulate the release of steroid gonadal hormones. After this initial response (flare effect), continuous exposure to GnRH leads to pituitary desensitization to GnRH with internalization of GnRH receptors and inactivation of the associated signaling cascade. This results in decreasing secretion of gonadotropins and ultimately sex steroids.[7]

Through this pathway, reproductive upregulation is suppressed, potentially providing a first-line therapeutic option for both the prevention and treatment of reproductive-associated hyperlipidemia in female birds. However, the specific lipid-lowering effects of deslorelin resulting from reproductive suppression have not been fully demonstrated or characterized in birds. In addition, although deslorelin implants have been studied in several avian species from different orders, blood lipids or other biochemistry analytes were not measured as part of these studies.[8–14] Because reproductive hyperlipidemia likely represents a significant percentage of psittacine bird dyslipidemia cases and is associated with reproductive disorders for which deslorelin implants are frequently prescribed by avian veterinarians, further investigation into the hypolipidemic effects of deslorelin implants in female birds is warranted.

Deslorelin acetate implants have been used off-label in avian medicine for the medical management of various reproductive disorders, including chronic egg laying, oviductal disease, and ovarian disease.[8,15,16] Deslorelin implants are currently labeled in the United States only for the treatment of ferret adrenal gland disease with an indexed veterinary product (Suprelorin F, Virbac, Fort Worth, TX, USA), for which extralabel use is prohibited by the US Food and Drug Administration Center for Veterinary Medicine.[17] Therefore, special authorization needs to be obtained to import a different product from another country that is not considered an indexed veterinary drug in the United States. Regulations are different in other countries where the extralabel use of specific deslorelin implant products is not necessarily prohibited in companion birds.

Cockatiels (*Nymphicus hollandicus*) represent a good research model for reproductive-associated lipid disorders due to the high prevalence of lipid-accumulation disorders and reproductive-induced hyperlipidemia in this species.[3,4] Additionally, there is available background research on the effects of deslorelin implants in cockatiels that has demonstrated their efficacy in suppressing egg production, with treated birds showing no egg production for 6 months.[8] The effect of deslorelin implants on egg laying and sex hormone blood concentrations has also been studied in multiple avian species, including Japanese quail (*Coturnix japonica*),[9,13,14] chickens (*Gallus gallus domesticus*),[18] pigeons (*Columba livia*),[10] quaker parrots (*Myiopsitta monachus*),[11] and lovebirds (*Agapornis* spp).[12]

The objective of this study was to evaluate the effects of a 4.7-mg deslorelin acetate subcutaneous implant on the blood lipid profile, including VLDL, intermediate-density lipoprotein (IDL), low-density lipoprotein (LDL), high-density lipoprotein (HDL), cholesterol, and triglycerides, and 31 steroid hormones and hormone precursors in a research colony of female cockatiels. The authors' hypothesis was that deslorelin implantation would result in a significant reduction in blood lipid concentrations for a shorter duration of time than observed for egg laying in previous studies, consistent with clinical observations by the authors.

MATERIALS AND METHODS

Animals

Sixteen female cockatiels from the University of California, Davis (Davis, CA, USA), cockatiel research colony were used for this study. The sample size was determined based on data available on cholesterol biological variation in this species and the ability to detect a difference of 10% in plasma cholesterol between treatments over time factoring a power of 80% and α of 5%.

Eleven cockatiels were 2 years old, 1 was 5 years old, 2 were 7 years old, and 2 were 8 years old, and they were considered healthy based on a recent physical examination. Females used in this research were not housed with males in the same cage, but males were present in the same room. The mean $\pm$ SD body weight was 93.0 $\pm$ 7.8 g. Cockatiels were offered pellets (Maintenance diet, Roudybush Inc, Woodland, CA, USA) and water ad libitum. All birds were exposed to a photoperiod of 12 hours of light and 12 hours of darkness, and the temperature of the room was maintained at approximately 24°C (75.2°F). The study was approved

by the University of California, Davis Institutional Animal Care and Use Committee (protocol 23491).

Experimental design

A complete randomized design was used for this study and the 16 female cockatiels were randomized into 2 groups of 8 birds, a group receiving deslorelin and a sham-procedure group, by a statistical software program (Version 4.3.2, 2024, R Foundation for Statistical Computing, Vienna, Austria). Randomization was done irrespective of individual pairing or cages.

The subject cockatiels were fasted overnight prior to the start of the study. The following morning, each cockatiel was manually restrained and approximately 0.8–1 mL of blood was collected from the right jugular vein with a 26-gauge needle and a 1-mL syringe. Blood was placed in EDTA pediatric collection tubes without plasma separator (Microtainer, BD, Franklin Lakes, NJ, USA), kept over ice, and centrifuged for 6 minutes at 3500*g* within 4 hours of collection. Plasma was then transferred into labeled 0.5-mL Eppendorf tubes and stored at −80°C (−112°F) until the samples were analyzed. After initial blood sampling, birds were sedated with a combination of midazolam 3 mg/kg IM (Midazolam, Hospira, Lake Forest, IL, USA) and butorphanol 2 mg/kg IM (Torbugesic, Zoetis Inc, Parsippany, NJ, USA). After 5 minutes, the interscapular skin was prepped with alcohol and a 4.7-mg deslorelin acetate implant (Suprelorin F 4.7 mg) was inserted under the skin with the supplied implanting needle and actuator syringe. Because sham implants were not available, the control group received a sham procedure inserting and removing one-third of a 16-gauge needle instead. A drop of cyanoacrylate tissue glue (3M Vetbond, 3M, St Paul, MN, USA) was used to close the skin. The sedation was reversed with flumazenil 0.1 mg/kg IM (Flumazenil, West-Ward, Eatontown, NJ, USA). Blood samples were then subsequently collected at 1, 2, 4, and 6 months after an overnight fast. Sampling was performed in the morning between 8 and 10 AM. At each time point, the implant was verified to still be present under the skin.

Lipid and lipoprotein analysis

Advanced lipoprotein profiling was performed on EDTA plasma with a high-resolution polyacrylamide gel electrophoresis kit (LDL Lipoprint kit, Quantimetrix, Redondo Beach, CA, USA) according to the manufacturer instructions with modifications for avian samples. Briefly, 25 µL of plasma was mixed with 200 µL of liquid loading gel containing Sudan black lipid dye and added to the top of precast 3% polyacrylamide gel tubes. After photopolymerization at room temperature for 30 minutes, samples were electrophoresed for 1 hour. The tubes were then scanned and the images analyzed for peak recognition and densitometry using a software program. As avian lipoproteins migrate at different locations along the gel compared with humans and mainly consist of HDL, the analysis of the gel was modified using a different software (GelAnalyzer 23.1, www.gelanalyzer.com) than the Lipoprint software (Lipoware, Quantimetrix, Redondo Beach, CA, USA) to identify and quantify VLDL, IDL, LDL, and HDL peaks. Relative concentrations were obtained in percentages of total area under the curve. Absolute concentrations were obtained based on plasma total cholesterol concentrations. Plasma total cholesterol and triglyceride concentrations were obtained with a reference laboratory analyzer (Roche Integra 400 Plus, Roche Diagnostics, Indianapolis, IN, USA) at the UC Davis Comparative Pathology Laboratory. Calculated values were also obtained and included non–HDL-C and HDL to total cholesterol ratio.

Steroid hormone analysis

Plasma EDTA samples were submitted on dry ice to the UC Davis West Coast Metabolomics Center for a targeted steroid panel including 31 steroid hormones and hormone precursors. Steroids were quantified by liquid chromatography–tandem mass spectrometry and included estriol, cortisone, cortisol, corticosterone, aldosterone, allo-pregnanolone, β-pregnanolone, estradiol, cortisol 21-sulfate, cortexolone, dehydroepiandrosterone, 2-methoxyestradiol, testosterone, testosterone glucuronide, estrone, etiocholanolone, etiocholanolone glucosiduronate, cis-androsterone, epiandrosterone, cortexone, androstanediol, 5-androstenediol, dihydrotestosterone, 17α-hydroxyprogesterone, 20α-dihydroprogesterone, androstenedione, 17-OH pregnenolone, progesterone, dehydroepiandrosterone sulfate, 7α-hydroxycholesterol, and 7-oxo-cholesterol.

Steroids were extracted from plasma with an antioxidant solution (0.2 mg/mL butylated hydroxytoluene/EDTA in 1:1 methanol water) via homogenization with a GenoGrinder (SPEX SamplePrep, Metuchen, NJ, USA) (2 × 30 seconds), followed by centrifugation. The supernatant was washed with appropriate solvents and lyophilized. Extracts were reconstituted in 100 µL of a methanol acetonitrile (50:50) solution containing 1 µM 1-phenyl-3-hexadecanoic acid urea and 1-cyclohexylureido-3-dodecanoic acid. Reconstituted samples were mixed, sonicated for 5 minutes, and centrifuged through spin filters, and the supernatant was transferred to glass inserts in high-performance liquid chromatography vials. Tritiated steroids were included

to monitor retention times, and deuterated internal standards enabled quantification. Samples were analyzed with a ThermoFisher Vanquish UPLC system coupled with a Thermo TSQ Altis mass spectrometer (Waltham, MA, USA). Steroid metabolite concentrations were quantified by dividing the sample peak area by the area of the corresponding deuterated internal standard and expressed as nanograms per milliliter in plasma.

Statistical analysis

Linear mixed models were used to assess the effects of treatment groups (deslorelin, control), time, and treatment × time on blood lipids and steroids with individual cockatiels as the random effect. Time was treated as an ordinal factor so that we could perform post hoc analysis between time points. Assumptions of linearity, homoscedasticity, and absence of outliers were assessed on residual plots and residuals quantile plots. If residuals were not normally distributed, the response was log transformed and normality was rechecked. Correlation over time was also checked by computing the autocorrelation function. Post hoc tests were performed by analysis of variance on the fixed effects with a Tukey adjustment. The proportion of individual birds that were hypertriglyceridemic (arbitrarily defined as >300 mg/dL) or hypercholesterolemic (arbitrarily defined as >350 mg/dL) over the 6-month monitoring period were compared between treatment groups by Fisher exact tests (excluding T0). R (version 4.3.2) was used for statistical analysis and GGPLOT2 for plots.[19] An α of 0.05 was used for statistical significance.

RESULTS

Implants were well tolerated by all cockatiels and no cockatiel removed an implant during the study. An outlying data point displaying extreme hyperlipidemia with triglycerides at 6492 mg/dL and cholesterol at 886 mg/dL (deslorelin group at 6 months) was excluded from linear mixed-model analysis. A plasma sample from 1 bird in the control group at the 4-month time point was inadvertently lost during processing or storage. No significant differences were seen at baseline (T0) between groups for the different analyzed lipids.

Triglycerides were log transformed for the statistical model. There was a significant treatment × time effect ($P = 0.016$). On post hoc analysis, cockatiels in the deslorelin group had significantly lower triglycerides than controls only at 1 month post–implant administration ($P = 0.025$) (Fig 1).

Cholesterol was also log transformed. There was a significant treatment × time effect ($P = 0.036$). On post hoc analysis, cockatiels in the deslorelin group had significantly lower total cholesterol than controls only at 1 month ($P = 0.011$). Although the total cholesterol also trended lower at 2 months, this difference was not significant at the 0.05 level of significance, but was significant at the 0.1 level ($P = 0.083$) (Fig 1).

There were significant differences in IDL-C and HDL-C between groups over time (treatment × time interaction effect, $P = 0.027$ and $P = 0.002$, respectively). Only HDL-C had to be log transformed to fulfill model assumptions. On post hoc analysis, cockatiels in the deslorelin group had significantly lower IDL-C and HDL-C than controls at 1 month ($P = 0.002$ and $P = 0.003$, respectively) and just lower IDL-C at 2 months ($P = 0.017$) (Fig 1).

Non–HDL-C values were also lower in the deslorelin group ($P = 0.018$), but because the treatment × time interaction effect was not significant ($P = 0.49$), a more precise duration of effect could not be given. No statistically significant differences were detected in VLDL-C, LDL-C, and total cholesterol to HDL ratio (all $P > 0.05$) (Fig 1).

Four of 8 cockatiels (50%) in the deslorelin group were found to be either hypercholesterolemic or hypertriglyceridemic at least once over the 6-month treatment period compared with all the cockatiels (8 of 8, 100%) in the control group, which was statistically significant ($P = 0.026$). Occurrence of hyperlipidemia was not clustered at any particular time points within treatments.

For steroid hormones, only 8 hormones/precursors had consistent measured concentrations above detection limits, including estriol, estradiol, progesterone, cortisone, corticosterone, etiocholanolone glucosiduronate, 7α-hydroxy-cholesterol, and 7-oxo-cholesterol. Only progesterone showed significant differences with treatment ($P = 0.02$), but the treatment × time interaction term was not significant ($P = 0.74$). Progesterone was log transformed and a heteroscedastic model was used for analysis because the variance in the nontreated group was significantly higher than in the treated group ($P < 0.001$) (Fig 2). Overall, progesterone was lower in the deslorelin treatment group than in controls and the plot suggested the effect lasted at least several months. Estradiol decreased at 1 month post–deslorelin implantation, but this decrease was not statistically significant (Fig 2). In addition, plasma triglyceride concentrations were strongly associated with plasma progesterone concentrations ($P < 0.001$), but not with other steroid hormones including estradiol ($P = 0.43$). Plasma cholesterol concentrations were not significantly associated with plasma steroid hormone concentrations (all $P > 0.05$).

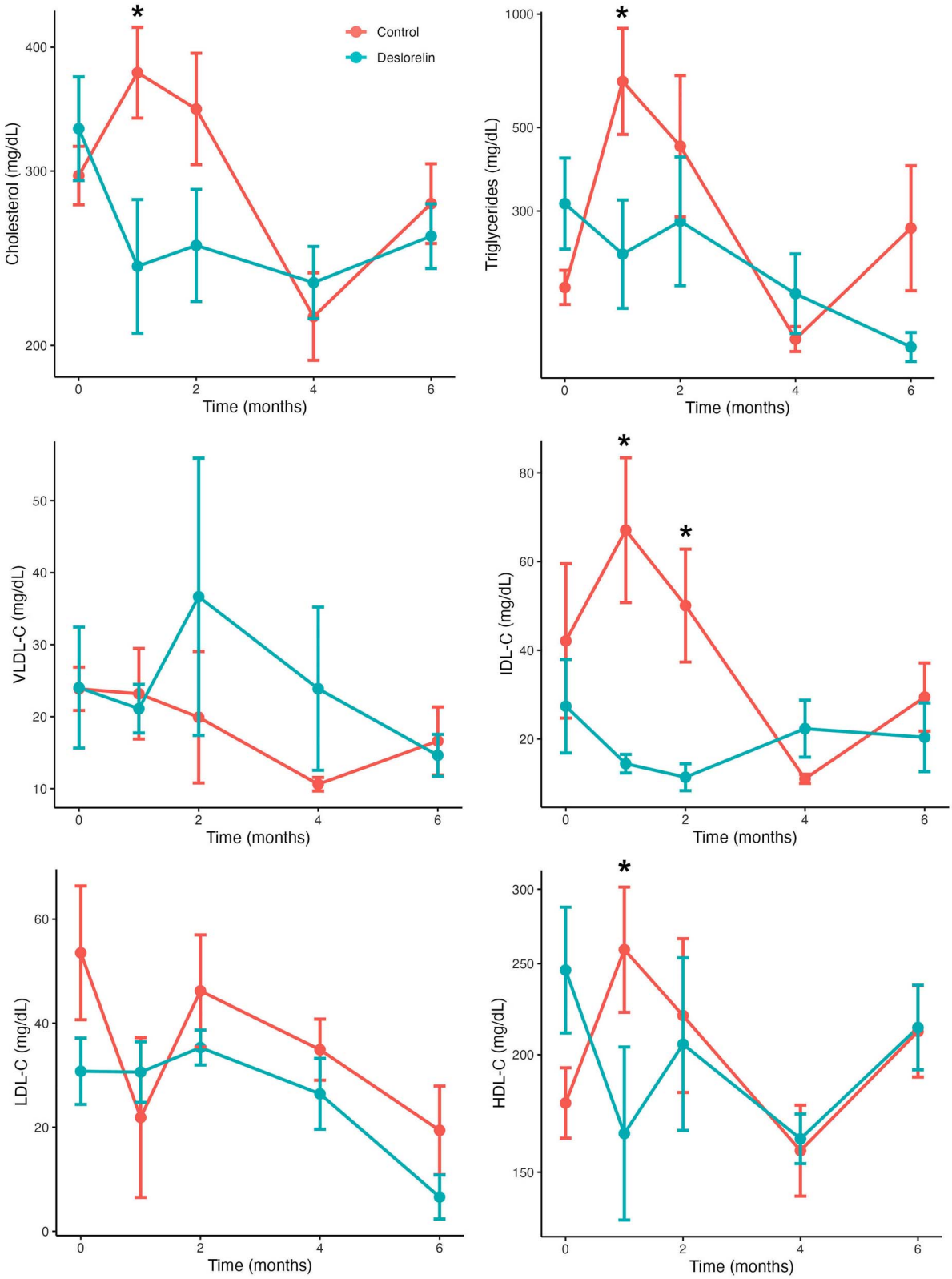

Figure 1. Mean ± SEM plasma concentrations of cholesterol, triglycerides, and lipoproteins over 6 months in 16 cockatiels (*Nymphicus hollandicus*) divided into a group that received a 4.7-mg deslorelin acetate implant at T0 (n = 8) and a control group (n = 8). Cholesterol, triglycerides, and HDL-C y-axes are on a log scale. *Significant differences between treatment groups ($P < 0.05$). One outlier data point was removed from analysis (individual from deslorelin group at 6 months). Abbreviations:VLDL-C, very low-density lipoprotein; IDL-C, intermediate-density lipoprotein; LDL-C, low-density lipoprotein; and HDL-C, high-density lipoprotein.

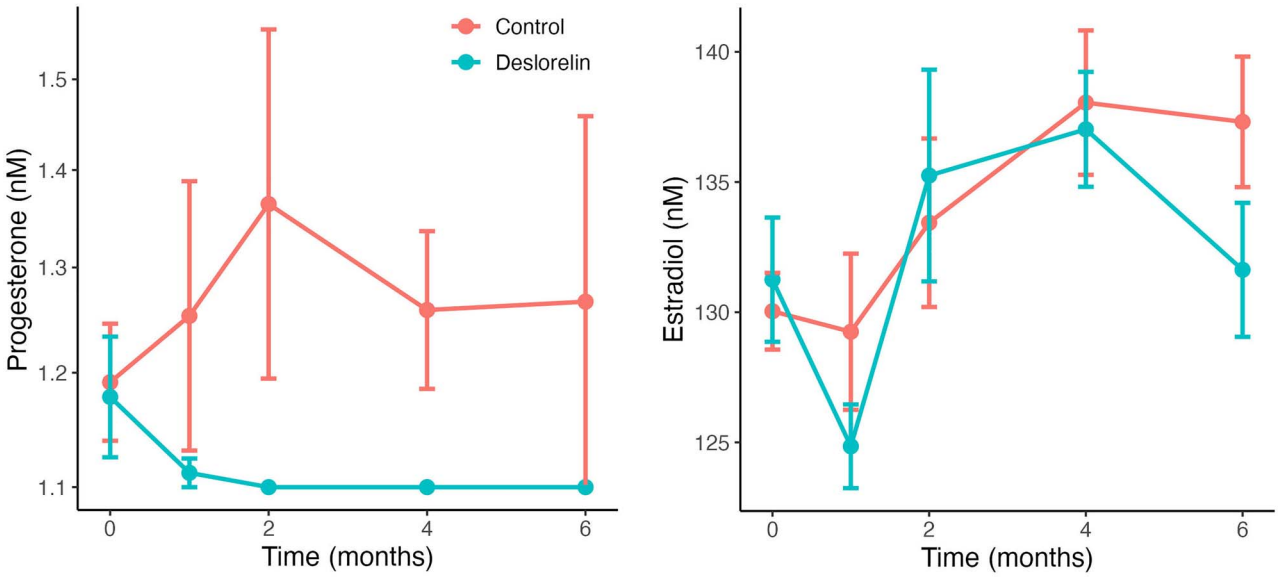

Figure 2. Mean ± SEM plasma concentrations of estradiol and progesterone over 6 months in 16 cockatiels (*Nymphicus hollandicus*) divided into a group that received a 4.7-mg deslorelin acetate implant at T0 and a control group. Progesterone concentrations were lower in the deslorelin group. Although estradiol decreased at 1 month, it was not significantly different from the control. Abbreviation: SEM, standard error of the mean.

DISCUSSION

As initially hypothesized, the deslorelin implants in the treatment group resulted in a significant reduction in blood lipid concentrations in cockatiels. However, the hypolipidemic effect was short-lived when compared with the reported effects of 6-month suppression of egg laying in this species[8]: it lasted for only 1 month for cholesterol, triglycerides, and HDL, and persisted for only approximately 2 months for IDL. Conversely, deslorelin resulted in a sharp decrease in the likelihood of a cockatiel being hyperlipidemic during the 6-month study period.

The observed decrease in plasma cholesterol and triglycerides seem to be related to a decrease in IDL and HDL, as characterized by high-resolution polyacrylamide gel electrophoresis. Intermediate-density lipoproteins are transitional lipoprotein particles that are triglyceride rich and sometimes referred to as VLDL remnants; they form as a result of the progressive lipolysis of VLDL by lipoprotein lipase. Surprisingly, the VLDL fraction, which is expected to contain VLDLy, did not change over time. In chickens, VLDLys are smaller than regular VLDLs,[6,20] but this is not necessarily true in all birds.[3] Vitellogenin is a phospholipid- and protein-rich lipoprotein that also has very high density due to its high protein content. In our study, the expected migration positions of VLDL and vitellogenin on the lipoprotein gel electrophoretic system were unknown. Also, as cholesterol is mainly carried as HDL in birds, a decrease in HDL is expected alongside a decrease in total cholesterol.

The duration of action of deslorelin implants on the inhibition of reproductive activity of female birds has been shown to be variable among species and was primarily demonstrated based on egg laying. In this study, using hyperlipidemia as a different endpoint, deslorelin was found to have a much shorter duration of action of about 1–2 months. In contrast, a study on 4.7-mg deslorelin implants in cockatiels found that egg laying was suppressed for 6 months.[6] In quaker parrots, a 4.7-mg deslorelin implant also limited reproduction and egg laying for 6 months.[11] Similarly, in Japanese quail, a 4.7-mg deslorelin implant reduced mean egg production for 3 months in 1 study,[9] and for at least 1.5 months in another.[13] In pigeons, deslorelin effectively controlled egg laying for at least 1.6 months.[10] Birds possess 3 distinct GnRH molecules,[21] which may contribute to inconsistencies in their response to GnRH superagonists. Compared with mammals, birds have shown a higher failure rate with these treatments, likely due to differences in their GnRH molecular structure.[22] Other external factors may also influence the variability in response in birds, such as species-specific reproductive physiology and endocrinology, husbandry, individual reproductive status at the time of implant application, and photoperiod, among others.

Surprisingly, of all the measured hormones, only plasma progesterone concentrations were affected by the deslorelin treatment, with minor effects on other

steroid hormones; a nonsignificant decrease in estradiol was seen at the 1-month time point. A decrease in plasma progesterone was also reported in lovebirds implanted with deslorelin, but reported data were limited because only an abstract was published from this study.[12] In quail, 4.7-mg deslorelin implants resulted in a significant decrease in estradiol at 1 month in most birds and for a longer duration of up to 3 months when excluding birds that continued to lay eggs.[9] Progesterone was not measured in that study. In a follow-up study with quail as subject animals and with double the deslorelin implant dose, progesterone was measured and found to be unaffected by the use of deslorelin implants.[14] In pigeons, deslorelin implantation was associated with a prolonged decrease in plasma LH, but progesterone and estradiol were not measured.[10] In addition, only progesterone concentrations were significantly associated with blood lipid concentrations in this study. These findings are intriguing because estradiol, rather than progesterone, is known to trigger vitellogenesis and reproductive hepatic lipogenesis.[2,5] Vitellogenesis in oviparous vertebrates, including birds, is a hormone-driven process in which estrogens play a key role in lipid and glucose metabolism as well as on the production of yolk and eggshell proteins.[2,23] Progesterone, synthesized by the granulosa cells of preovulatory follicles, plays a minimal role in vitellogenesis. Its circulating concentrations primarily rise prior to ovulation, inducing a preovulatory LH surge through a positive feedback mechanism that leads to ovulation.[5] Exogenous administration of estradiol to chicken hens consistently results in marked increase in plasma lipids, mainly triglycerides, cholesterol, and phospholipids. Conversely, exogenous administration of progesterone typically decreases plasma lipids or has minimal effect in chickens.[24] In addition, restricted-ovulation chickens, which are hyperlipidemic, have high estradiol and low progesterone when compared with standard chickens.[25] Nevertheless, the temporal pattern of hypolipidemic effects observed in this study mirrored the fluctuation seen with estradiol concentrations, with a dip at 1 month that was followed by a rebound, although this trend did not reach statistical significance. It is also possible that a more pronounced decline in estradiol occurred before the 1-month time point but went undetected due to the absence of plasma measurements between implantation and the 1-month mark. The concurrent decline in progesterone is likely attributable to reduced concentrations of gonadotropins and the resorption of follicles. The positive association between low progesterone concentrations and decreasing plasma lipids is perplexing and hard to interpret in light of what is known on avian reproductive biology and vitellogenesis hormonal control, but it could be circumstantial or related to other factors occurring concurrently as a result of deslorelin treatment. Furthermore, chickens are continuous undeterminate layers that have been selected over long periods of time for increased and constant egg production. Therefore, the hormonal control of vitellogenesis in other birds, such as Psittaciformes, which are seasonal breeders and mostly determinate layers, may show differences.

The main limitation of this study is that, although many female birds showed evidence of reproductive activity, these birds were considered healthy and not suffering from persistent or dysregulated vitellogenesis, so they may have been at different stages of vitellogenesis and follicular development. A clinical trial performed on birds diagnosed in a clinical setting with persistent reproductive hyperlipidemia due to upregulated/dysregulated vitellogenesis and reproductive disorders might show different results, and it is possible that the hypolipidemic effects of deslorelin on these birds would be more long-lasting. Having an ability to directly measure reproductive lipoproteins (vitellogenin and VLDLy) would also be helpful to better characterize the effects of deslorelin in female birds.

This study provides evidence of the potential of deslorelin to modulate lipid metabolism in female cockatiels and suggests a temporal effect lasting 1–2 months. Our results provide novel insights into the metabolic effects of deslorelin beyond its reproductive role, and indicate that it may have implications for managing hyperlipidemia in avian species. Further studies are needed to characterize circulating hormonal response to deslorelin implantation and its relationship with vitellogenic blood lipid dynamics.

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