Intergenerational effects of maternal growth strategies in broiler breeders

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ABSTRACT Maternal growth patterns affect broiler growth performance. The current study investigated the impact of lesser growth restriction, compared to the breeder-recommended target growth, during the prepubertal growth phase and earlier pubertal growth in breeders on their offspring growth and carcass traits. In a randomized controlled trial, a total of 40 female broiler breeders were randomly assigned to 10 unique growth trajectories with 2 levels of maternal BW gain (**MW**) in prepubertal phase and 5 levels of maternal pubertal growth inflection (**MI**) for each level of the MW. Growth parameters (MW and MI) were estimated by fitting a 3-phase Gompertz model to the breeder-recommended BW target (Standard MW; **SMW**), or 10% higher (**HMW**). Maternal pubertal inflection was advanced by 0, 5, 10, 15, or 20% in both SMW and HMW groups. Maternal growth trajectories were implemented from 0 to 42 wk of age using a precision feeding (**PF**) system. The current study consisted of two cohorts that varied in maternal age (**MA**) of 35 and 42 wk. The broiler chicks were fed to 35 d of age, also with the PF system. Analysis of covariance was conducted on all dependent variables (BW, FCR, carcass traits) with MA, MW, and offspring sex as categorical variables and MI as a continuous predictor variable. Chicks from 42 wk old hens had higher 0 (hatch), 14, 21, and 28 d BW, liver, and heart weight, and lower FCR from 7 to 35 d of age than those from the 35 wk old hens. Compared to SMW hens, HMW hens produced female offspring with lower FCR, and male offspring with heavier gut weight. Advancing MI increased hatch BW in both sexes and 35 d BW in male broilers. For every week that the MI was advanced, hatch BW increased by 0.26 g in females and 0.39 g in males; however, 21 and 35 d BW decreased by 6.85 and 17.29 g/wk in females and increased by 10.53 and 25.94 g/wk in males, respectively. Overall, a lesser degree of growth restriction during prepubertal and earlier pubertal growth increased male offspring growth.

Key words: broiler breeder, carcass, feed restriction, intergenerational, multi-phasic growth

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INTRODUCTION

Controlling body weight in broiler breeder farms is achieved through feed restriction. The gap between growth potential of broilers and broiler breeder target BW has increased over the last 60 yr (Renema et al., 2007). Thus, the intensity of broiler breeder feed restriction has increased which can impair reproductive performance (van der Klein et al., 2018; Zuidhof, 2018) and raise welfare concerns (van Krimpen and de Jong, 2014). The degree of feed restriction depends on the target growth curve; optimality of primary breeder-prescribed growth curves has rarely been reported. It is also valuable to investigate the intergenerational impact of lesser growth restriction and earlier pubertal growth.

Broiler growth rate, body composition, feed intake level, and skeletal health status are highly affected by their genetic potential (Havenstein et al., 2003). Breeder management practices, maternal age and maternal nutrition have also been reported to affect broiler performance (Triyuwanta et al., 1992; Kidd, 2003; Calini and Sirri, 2007; Enting et al., 2007). Most of the research pertaining to consequences of maternal effects in chickens have focused on nutrient composition of the diet; however, there is little data on effects of alterations of the maternal prepubertal BW gain (**MW**) and pubertal inflection (**MI**) on progeny performance in the literature. It has been reported that increasing target BW and the amount of feed available to broiler breeders increased offspring's hatch BW (van der Waaij et al., 2011) and final BW (van der Waaij et al., 2011; van Emous et al., 2015; Bowling et al., 2018).

Maternal feed restriction intensity can affect offspring abdominal fat deposition. van der Waaij et al. (2011) found that offspring of feed restricted breeders had significantly lower BW and relatively more abdominal fat deposition compared to those of breeders fed ad libitum.

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They concluded that it might be due to a mismatch between maternal and offspring feeding levels and nutritional environment which would potentially lead to economic loss and impaired feed efficiency. Humphreys (2020) observed heavier gut weight in broilers from 40-wk old hens that weighed 121% of the standard target BW compared to those of from standard BW hens.

The objective of the current study was to investigate the effect of a reduced degree of maternal prepubertal phase growth restriction and earlier maternal pubertal phase growth on offspring growth and development. It was hypothesized that increased MW and advanced MI would increase progeny hatch BW, final BW, and digestive tract weight, and lower MW would increase fat pad weight.

MATERIALS AND METHODS

The animal protocol for the study was approved by the University of Alberta Animal Care and Use Committee for Livestock and followed the Canadian Council on Animal Care Guidelines and Policies (CCAC, 2009).

Maternal Study Design

In a randomized controlled trial, a total of 40 female Ross-708 broiler breeder pullets were randomly assigned to 10 growth trajectories (Figure 1) that were implemented using a precision feeding (**PF**) system. The maternal growth trajectories were designed using a 3-phase Gompertz model fit to the breeder-recommended target BW. The model had the form (Zuidhof, 2020):

$$BWt = \sum_{i=1}^{i=3} g_i exp^{-exp^{-b_i(t-I_i)}} + \varepsilon_t$$

where BW_t was BW (kg) at time t (wk); g_i was the total amount of gain (kg) accruing in phase i; b_i was the growth rate coefficient; t was age (wk); I_i was the inflection point (wk), or the age at which growth for phase i reached its maximum rate; and ε_t was the residual error with an expected value of 0, and a normally distributed variance estimated by the software $\varepsilon_t \sim N(0,SD^2)$; i was the growth phase (i = 1 to 3) where phase 1, 2, and 3 corresponded to prepubertal, pubertal, and postpubertal growth phases. The maternal growth trajectories were designed with 2 levels of g_i in prepubertal phase as discrete variables; g_1 was either the estimated gain for phase 1 derived from the breeder-recommended standard BW (**SMW**) target, or 10% higher (**HMW**). The coefficient I_2, which biologically defined the inflection point of the pubertal growth phase, was advanced by 0, 5, 10, 15, or 20% of the coefficient estimated when fitting to the breeder-recommended target BW. I_2 was a continuous variable imposed in both the SMW and HMW groups. Each bird was an experimental unit.

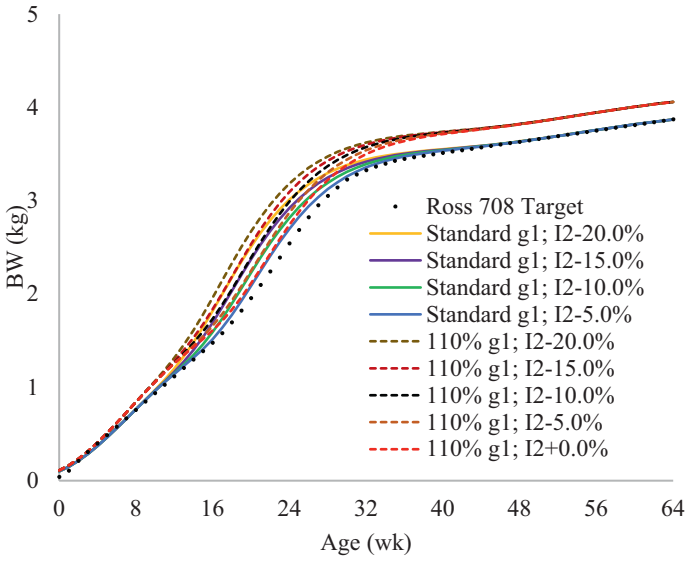

Figure 1. Fit of a 3-phasic Gompertz model to target BW of Ross 708 broiler breeders (. . .) and increased total amount of gain (kg) in prepubertal growth phase (g_1) by 10% (—) with earlier pubertal growth inflection time (I_2) at 0, 5, 10, 15, and 20% of the standard BW curve.

Parent Stocks and Management

The pullets were housed in a single pen containing 2 PF stations, from hatch to 43 wk of age at a stocking density of 3.0 birds per m^2. Water was provided ad libitum throughout the experiment. They were fed commercial diets: starter (crumble; AME 2,726 kcal/kg, 21% CP, 1.00% Ca, and 0.45% available P) from hatch to d 34; grower (mash; AME 2,799 kcal/kg, CP 15%, 0.79% Ca, and 0.44% available P) from d 35 to d 179; and laying diet (crumble; AME 2,798 kcal/kg, 15.3% CP, 3.30% Ca, and 0.38% available P) from 180 d onward. All birds were fed individually using a PF system (Zuidhof et al., 2019) that permitted feed intake levels appropriate to achieve the target growth trajectories of each individual bird. At 14 d of age each bird was equipped with a wing band containing a radio frequency identification (RFID) transponder to be recognized individually by the PF system. The PF system provided access to a meal based on the individual preprogrammed target BW. If the BW exceeded the target BW, the system gently ejected the birds from the PF station. The birds had access to the PF system 24 h per day throughout the experiment. Throughout the experiment, every time a bird entered the feeding station, its RFID, real-time BW and ADFI data (if fed) were recorded to a PF system database. Daily median BW for each individual bird were determined from database records of all visits to the PF station.

Settable eggs were collected from the experimental hens at 35 (cohort 1) and 42 wk of age (cohort 2) over 7 d prior incubation to conduct two separate offspring cohorts. These eggs were identified by hen and date, stored at 16°C and set into single-stage incubators with a randomized location. At 18 d of incubation, eggs were transferred to individual chick-hatching compartments with a newly randomized tray position.

Egg Components

Eggs were collected from every hen 1 wk prior to cohort 1 (236 to 241 d) and cohort 2 (282 to 287 d) and

immediately were used for egg proportion analysis. Eggs were separated into yolk, albumen, and shell. Dry weight of each component was determined after placing them into drying oven at 60°C for 4 days.

Broiler Study Experimental Design

The current progeny broiler study was designed as a completely randomized and controlled experiment. It included 2 replicated experiments that differed in maternal age (35 and 42 wk of age, which were called cohort 1 and 2, respectively). The experimental treatments were 10 unique maternal growth trajectories applied to the broiler breeders. Broilers were fed individually using the PF system. Therefore, each bird was an experimental unit.

Broiler Stocks and Management

Two offspring cohorts were conducted that differed in maternal age (**MA**): 35 and 42 wk of age. At hatch, chicks were feather-sexed, weighed, and identified with bar-coded neck tags (Heartland Animal Health Inc., Fair Play, MO). A total of 124 chicks (on average 12 chicks per maternal treatment) from each maternal age were randomly placed in environmentally controlled pens (n = 4) to 35 d of age. The initial set temperature was 32°C, which decreased 1°C every 3 d until 22°C. The photoperiod was 23L:1D (16 lx) from d 0 to 3 and decreased by 1 h of light each day until d 7 where the photoperiod remained at 19L:5D (8 lx) for the duration of the experiment. Wheat-corn-soybean-based diets were provided ad libitum in pelleted form as follows: starter (3,044 kcal of ME/kg; 23% CP; 1.27% Lys) from 0 to 11 d; grower (3,091 kcal of ME/kg; 22% CP; 1.18% Lys) from 11 to 21 d; and finisher (3,170 kcal of ME/kg; 21% CP; 1.13% Lys) from 21 to 40 d. Similar to the parent stock, the PF system recorded RFID, BW and FI data throughout the cohorts. All PF stations were turned off 12 h prior to euthanasia to achieve an empty gut weight. At 35 d of age, all broiler chicks were humanely euthanized and dissected. Breast muscle (pectoralis major and pectoralis minor), abdominal fat pad (including fat removed from the gizzard), liver, heart, and gastrointestinal tract (gut; 1 cm above the crop to the end of colon, adhering fat removed from the gizzard) weights were recorded.

Statistical Analysis

Analysis of covariance was conducted on all dependent variables using the MIXED procedure of SAS (Version 9.4, SAS Institute Inc., Cary, NC), with broiler sex, maternal age and MW as sources of variation, MI as a continuous predictor variable, and dam as a random subject. In addition, one- and 2-way ANOVA were conducted using the MIXED procedure of SAS respectively on egg weight (**EW**) and maternal median BW (**MMBW**) during the period of egg collection for each progeny cohort at 35 and 42 wk of age to determine the relationship between EW and MMBW, and MI and MMBW. Pairwise differences between means were determined using Tukey's HSD test with the PDIFF option of the LSMEANS statement and were reported as different when $P \leq 0.05$. Trends were reported where $0.05 < P \leq 0.10$.

RESULTS AND DISCUSSION

Egg Components

Eggs from 41 wk of age hens were heavier than those from 34 wk of age ($P = 0.006$, Table 1). There was no effect of MW or MI on egg weight, dry eggshell, yolk, and albumen weight at either maternal age (Table 1). In the current study, trajectory-specific BW targets converged at 46 wk of age (Figure 1). Thus, target BW between BW trajectories differed more at 34 wk (a week prior to the egg collection period for the first cohort) compared to 41 wk (a week prior to the egg collection period for the second cohort). There was a negative relationship between MMBW and MI at 34 wk of age

Table 1. Effects of maternal prepubertal BW (MW) and maternal pubertal growth inflection (MI) on egg weight, dry eggshell, yolk, and albumen weight from 40 to 41 wk of age.

Effect	MA[1]	MW[2]	EW[3]	SEM	Eggshell	SEM	Egg yolk	SEM	Egg albumen	SEM
			g							
MW		S	61.78	0.56	5.51	0.04	9.51	0.07	4.53	0.03
		H	62.84	0.62	5.41	0.06	9.84	0.08	4.89	0.05
MA	35wk		61.11[b]	0.58	5.41	0.04	9.63	0.08	4.68	0.03
	42wk		63.51[a]	0.60	5.52	0.06	9.72	0.06	4.73	0.05
			g/wk							
MI			-0.43	0.35	0.003	0.022	-0.032	0.043	-0.029	0.021
MI × MW		S	-3.43	0.56	-0.56	0.89	0.44	1.24	-0.56	0.71
		H	-3.21	0.51	-0.53	0.044	0.43	0.062	-0.51	0.035
Source of variation			*P*-value							
MW			0.74		0.53		0.11		0.43	
MI			0.22		0.50		0.25		0.73	
MI x MW			0.67		0.61		0.72		0.20	
MA			0.006		0.12		0.42		0.37	

[a,b]LSMeans within column and effect lacking a common superscript differ ($P \leq 0.05$).
[1]MA: Maternal age
[2]MW: S = standard (breeder recommended) maternal prepubertal gain; H = maternal prepubertal BW gain 10% higher than the standard one.
[3]EW: Egg weight

Table 2. Effects of maternal prepubertal BW (MW), maternal pubertal growth inflection (MI), and offspring sex on BW at 0, 7, 14, 21, 28, and 35 d of broiler chickens.

				BW (g)											
Effect	MA[1]	MW[2]	Sex[3]	0 d	SEM	7 d	SEM	14 d	SEM	21 d	SEM	28 d	SEM	35 d	SEM
MW		S		42.3	0.4	150	1.7	356	5.0	746	11.8	1,300	21.5	1,916	31.3
		H		43.6	0.4	152	1.9	359	5.3	755	11.6	1,323	22.4	1,942	31.0
Sex			F	43.0	0.4	150	1.8	352	5.0	734[b]	11.5	1,274[b]	21.1	1,857[b]	27.6
			M	42.9	0.4	152	1.8	363	5.3	767[a]	11.7	1,349[a]	22.5	2,001[a]	34.4
MW × Sex		S	F	42.6	0.5	150	2.4	354	6.9	738	16.5	1,274	28.1	1,853[b]	35.9
			M	42.0	0.6	149	2.5	358	7.1	753	16.3	1,325	32.2	1,979[ab]	52.2
		H	F	43.5	0.6	150	2.7	349	7.2	730	16.3	1,274	31.5	1,862[ab]	41.7
			M	43.8	0.4	154	2.7	368	7.8	780	16.6	1,372	31.7	2,022[a]	45.6
MA	35wk			42.3[b]	0.4	151	1.8	325[b]	4.7	684[b]	10.9	1,216[b]	20.8	1,903	30.8
	42wk			43.6[a]	0.4	151	1.9	390[a]	5.8	817[a]	13.0	1,407[a]	23.7	1,955	31.4
				g/wk											
MI x Sex			F	-0.26	0.322	-1.02	1.58	2.74	4.59	6.85	10.80	12.45	18.62	17.29	23.91
			M	-0.39	0.48	-0.82	2.20	-3.40	6.17	-10.53	14.35	-12.39	26.61	-25.94	39.66
MI x MW		S		5.17	9.74	0.12	0.47	75.61	12.99	84.07	29.85	256.45	54.63	408.62	71.25
		H		4.95	0.48	0.11	2.35	71.60	6.50	79.49	14.90	243.68	27.31	388.72	35.56
Source of variation				*P*-value											
MW				0.73		0.95		0.37		0.34		0.16		0.16	
MI				0.035		0.26		0.31		0.21		0.18		0.097	
MI x MW				0.89		1.00		0.38		0.37		0.18		0.18	
Sex				0.90		0.93		0.15		0.023		0.035		0.017	
MW x Sex				0.66		0.95		0.94		0.59		0.46		0.50	
MI x Sex				0.92		0.90		0.18		0.033		0.054		0.031	
MI x MW x Sex				0.61		1.00		0.99		0.65		0.50		0.52	
MA				0.017		0.84		< 0.001		< 0.001		< 0.001		0.24	

[a,b]LSMeans within column and effect lacking a common superscript differ ($P \leq 0.05$).
[1]MA: Maternal age.
[2]MW: S = standard (breeder recommended) maternal prepubertal gain; H = maternal prepubertal BW gain 10% higher than the standard one.
[3]Sex: F = Female; M = Male.

(-24 g/wk of advanced MI) which reduced by 41 wk (-14 g/wk of advanced MI), because the target growth trajectories were converged by 46 wk of age (Figure 1). Therefore, increasing MW or advancing MI did not increase EW at those ages. In consistence with the current study previous research showed that increasing target BW by 8% at 20 wk of age (Fattori et al., 1991), 20% at 18 wk of age (Hocking et al., 2002) 8% at 20 wk of age (van Emous et al., 2013), 16 and 20% at 20 wk of age (Gous and Cherry, 2004; Ekmay et al., 2012) did not affect average egg weight. However, in other research implementing higher target BW by 21% (Renema et al., 2001a,b) and 13% (Sun and Coon, 2005) at 20 wk of age increased egg weight. In the current study however, MMBW was under the target BW in a hen causing a variation in MMBW inside the HMW treatment group. Thus, the effect of individual MMBW (regardless of the treatment) on EW of the eggs collected for each cohort was investigated. The results showed that increasing MMBW increased EW ($P = 0.001$), and MA tended to increase the EW ($P = 0.064$). It has been reported that egg size is an important factor in the chick weight, chick quality, and performance of broiler chicks to market weight (Abiola et al., 2008; Iqbal et al., 2016 and 2017) while others have found that any advantage of chicks hatched from large-sized eggs diminishes rapidly after hatching (Yannakopoulos and Tserveni-Gousi, 1987; Pinchasov, 1991).

BW and FCR

The chicks from 42 wk old breeders had higher 0 (hatch), 14, 21, and 28 d BW compared to those from 35 wk old hens (Table 2). Earlier MI increased hatch BW. For every week that the MI was advanced, hatch BW increased by 0.26 and 0.39 g in females and males, respectively. The effect of MI on BW at 21 and 35 d of age depended on sex (Figure 2). Specifically, males and females responded differently to MI. For every week that MI was advanced males 21, 28 and 35 d BW was increased by 10.53, 12.39 and 25.94 g, respectively. However, there was a 6.85, 12.45 and 17.29 g reduction in BW for females at those ages (Figure 2). When breeder-recommended target BW was increased by 121%, the final BW of offspring of HMW hens were 4% higher than those of SMW ones (Humphreys, 2020). In the current study, MW did not affect 35 d BW. Male broilers from breeders whose MI was advanced from 22 to 18 wk of age had greater BW on 21 and 35 d indicating a sex-dependent effect of MI on offspring BW. This may be related to sex-specific genetic potential, which affects body composition (Zuidhof, 2005), plasma hormone levels (Gonzales et al., 2003), and muscle development (Henry and Burke, 1998). Bowling et al. (2018) found that increasing dam BW by 15% increased male broiler BW by 8.5% than that of the standard group. The authors further found that the concentration of yolk corticosterone of low BW hens was 1.2 times greater than that of high BW hens and suggested that males may be more sensitive to maternal feed restriction-induced stress. It is possible that in the current study, sex-dependent differences in 35 d BW might have been due to the reduced stress as a result of earlier MI and concomitant relaxed levels of feed restriction during the maternal pubertal phase.

For every week that MI was advanced, ADFI decreased by 0.92 and 0.03 g/d in female and male broilers, respectively. Average daily feed intake of HMW and SMW offspring respectively decreased by 18.33 and 19.33 g/wk of advanced MI ($P = 0.040$, Table 3). It has been shown that offspring of feed restricted might have higher ADFI (van Emous et al., 2015 in broiler breeders; Vickers et al., 2000 in rats). It is possible that in the current study, low maternal ADFI in SMW dams may have triggered induced reprogramming of genes that are responsible for feed intake through an epigenetic effect at a lower level of their offspring (van der Waaij et al., 2011).

Feed conversion ratio decreased in the second week compared to the first week studied (Table 3). Digestive tract maturation and development from 7 to 10 d may have resulted in poor utilization of nutrients, thus increasing FCR (Batal and Parsons, 2002). In addition, birds were being trained to use the PF system individually from 7 to 14 d of age, which may have decreased their ability to conserve energy and their feed intake. FCR of chicks from older breeders (42 wk) was lower than that of

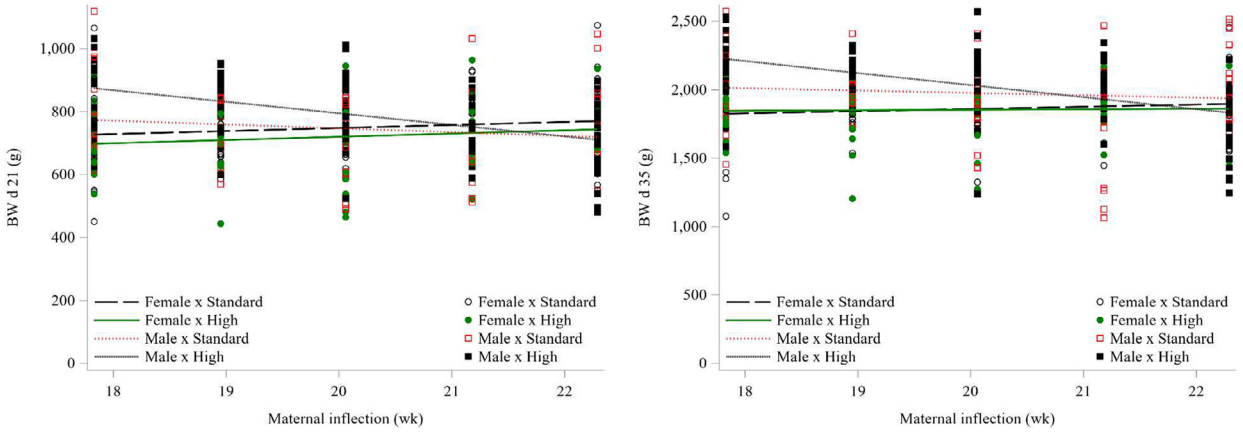

Figure 2. Effects of maternal prepubertal growth (MW), maternal pubertal growth inflection (MI), and offspring sex on 21 and 35 d BW of offspring broilers.

Table 3. Effects of maternal prepubertal BW (MW), maternal pubertal growth inflection (MI), and offspring sex on FCR and daily feed intake at different ages of broiler chickens.

				FCR (g/g)										ADFI (g/d)	
Effect	MA[1]	MW[2]	Sex[3]	7−14 d	SEM	14−21 d	SEM	21−28 d	SEM	28−35 d	SEM	7−35 d	SEM	7−35 d	SEM
				g/g										g/d	
MW		S		1.441	0.025	1.312	0.008	1.451	0.007	1.415	0.012	1.425	0.008	90.0	1.6
		H		1.448	0.027	1.322	0.009	1.450	0.007	1.425	0.010	1.430	0.007	91.1	1.5
Sex			F	1.467	0.027	1.329	0.008	1.459	0.007	1.427	0.011	1.441	0.007	87.9[b]	1.4
			M	1.422	0.025	1.305	0.008	1.442	0.007	1.414	0.011	1.414	0.008	93.2[a]	1.7
MW × Sex		S	F	1.460	0.032	1.326	0.010	1.461	0.010	1.435	0.016	1.444	0.010	88.2	1.9
			M	1.422	0.033	1.298	0.012	1.441	0.009	1.396	0.017	1.406	0.013	91.7	2.6
		H	F	1.474	0.037	1.332	0.013	1.457	0.010	1.418	0.015	1.437	0.010	87.6	1.9
			M	1.423	0.032	1.312	0.011	1.443	0.011	1.431	0.014	1.422	0.010	94.6	2.2
MA	35wk			1.641[a]	0.039	1.308	0.009	1.426[b]	0.008	1.448[a]	0.010	1.479[a]	0.009	92.6	1.5
	42wk			1.248[b]	0.016	1.326	0.008	1.475[a]	0.007	1.392[b]	0.012	1.376[b]	0.007	88.5	1.5
				g/g/wk										g/d/wk	
MI x Sex			F	-0.041	0.019	0.013	0.006	-0.0007	0.0068	-0.0051	0.0099	-0.0001	0.0062	0.92	1.2
			M	-0.029	0.026	0.005	0.009	-0.0025	0.008	0.016	0.013	0.021	0.009	0.03	2.04
MI x MW		S		-0.480	0.598	0.294	0.209	-0.073	0.191	0.0279	0.276	0.031	0.175	19.33	3.50
		H		-0.456	0.029	0.279	0.010	-0.069	0.009	0.0256	0.013	0.029	0.008	18.33	1.74
Source of variation															
MW				0.10		0.12		0.84		0.16		0.047		0.037	
MI				0.051		0.22		0.47		0.56		0.21		0.17	
MI x MW				0.098		0.14		0.83		0.18		0.051		0.040	
Sex				0.25		0.65		0.37		0.051		0.12		0.032	
MW x Sex				0.68		0.63		0.44		0.21		0.049		0.16	
MI x Sex				0.29		0.54		0.30		0.060		0.18		0.049	
MI x MW x Sex				0.70		0.61		0.46		0.27		0.059		0.18	
MA				<0.001		0.13		<0.001		0.002		<0.001		0.068	

[a,b]LSMeans within column and effect lacking a common superscript differ ($P \leq 0.05$).

[1]MA: Maternal age.

[2]MW: S = standard (breeder recommended) maternal prepubertal gain; H = maternal prepubertal BW gain 10% higher than the standard one.

[3]Sex: F = Female; M = Male.

Table 4. Effects of maternal prepubertal BW (MW), maternal pubertal growth inflection (MI), and offspring sex on individual breast, fat pad, liver, heart, and gastro-intestinal tract (GIT) weight.

Effect	MA[1]	MW[2]	Sex[3]	Breast	SEM	Fat pad	SEM	Liver	SEM	Heart	SEM	GIT	SEM
				(g)									
MW		S		407	9.0	23.9	0.8	35.0	0.6	11.0	0.3	119.9	1.6
		H		416	8.2	23.8	0.9	35.0	0.7	11.2	0.3	120.5	1.6
Sex			F	407	8.6	25.2	0.8	33.1	0.6	10.1	0.3	114.1[b]	1.4
			M	416	8.3	22.6	0.9	36.8	0.7	12.1	0.3	126.3[a]	1.8
MW × Sex		S	F	409	12.0	25.9	1.0	33.3	0.7	9.9	0.3	114.1[b]	1.7
			M	404	13.7	22.0	1.3	36.6	1.1	12.2	0.5	125.6[a]	2.7
		H	F	404	12.2	24.4	1.3	32.9	0.9	10.3	0.4	114.0[b]	2.2
			M	428	10.4	23.2	1.3	37.1	1.0	12.0	0.3	126.9[a]	2.3
MA	35wk			405	9.1	23.1	0.9	33.8[b]	0.6	10.6[b]	0.3	119.0	1.5
	42wk			417	8.3	24.7	0.8	36.2[a]	0.7	11.6[a]	0.3	121.4	1.7
				g/wk									
MI × Sex			F	6.12	7.94	-0.11	0.68	0.30	0.46	-0.06	0.22	-0.60	1.12
			M	-4.9	11.46	0.74	1.03	-0.04	0.76	-0.24	0.35	2.62	1.99
MI × MW		S		201.39	22.10	0.29	2.14	10.21	1.45	5.89	0.67	-39.12	3.54
		H		191.11	11.02	0.20	1.07	9.69	0.72	5.62	0.33	-37.18	1.77
Source of variation				*P*-value									
MW				0.071		0.20		0.057		0.45		0.55	
MI				0.11		0.62		0.15		0.047		0.39	
MI × MW				0.080		0.19		0.057		0.46		0.56	
Sex				0.058		0.95		0.058		0.45		0.93	
MW × Sex				0.62		0.21		0.31		0.65		0.048	
MI × Sex				0.067		0.92		0.11		0.72		0.73	
MI × MW × Sex				0.69		0.24		0.33		0.70		0.051	
MA				0.35		0.20		0.008		0.018		0.28	

[a,b]LSMeans within column and effect lacking a common superscript differ ($P \leq 0.05$).
[1]MA: Maternal age.
[2]MW: S = standard (breeder recommended) maternal prepubertal gain; H = maternal prepubertal BW gain 10% higher than the standard one.
[3]Sex: F = Female; M = Male.

Table 5. Effects of maternal prepubertal BW (MW), maternal pubertal growth inflection (MI), and offspring sex on individual breast, fat pad, liver, heart, and gastro-intestinal tract (GIT) as a percent of live BW.

Effect	MA[1]	MW[2]	Sex[3]	Breast	SEM	Fat pad	SEM	Liver	SEM	Heart	SEM	GIT	SEM
				(% of live BW)									
MW		S		21.09	0.21	1.23	0.03	1.82	0.02	0.58	0.01	6.39	0.12
		H		21.43	0.22	1.21	0.04	1.81	0.02	0.58	0.01	6.28	0.11
Sex			F	21.84	0.21	1.34	0.04	1.79	0.02	0.55	0.01	6.22	0.10
			M	20.69	0.22	1.11	0.04	1.84	0.02	0.61	0.01	6.45	0.13
MW × Sex		S	F	21.94	0.29	1.38	0.05	1.80	0.03	0.54	0.02	6.21	0.14
			M	20.24	0.31	1.09	0.05	1.85	0.04	0.61	0.02	6.56	0.21
		H	F	21.73	0.31	1.30	0.06	1.78	0.03	0.56	0.02	6.22	0.15
			M	21.13	0.32	1.12	0.06	1.83	0.03	0.60	0.02	6.33	0.17
MA	35wk			21.16	0.22	1.19	0.04	1.77[b]	0.02	0.56	0.01	6.34	0.10
	42wk			21.36	0.22	1.25	0.04	1.86[a]	0.02	0.60	0.01	6.32	0.14
				% of live BW/ wk									
MI × Sex			F	0.15	0.19	-0.007	0.031	0.004	0.017	-0.008	0.011	-0.08	0.09
			M	-0.01	0.26	0.044	0.042	0.017	0.027	-0.008	0.017	0.17	0.15
MI × MW		S		4.58	0.55	-0.155	0.096	0.120	0.052	0.218	0.033	-3.16	0.26
		H		4.35	0.27	-0.146	0.048	0.114	0.026	0.208	0.016	-3.01	0.13
Source of variation				*P*-value									
MW				0.19		0.33		0.48		0.65		0.28	
MI				0.63		0.94		0.70		0.34		0.071	
MI × MW				0.22		0.31		0.45		0.63		0.30	
Sex				0.58		0.44		0.87		0.33		0.074	
MW × Sex				0.89		0.23		0.69		0.18		0.69	
MI × Sex				0.40		0.67		0.77		0.23		0.058	
MI × MW × Sex				1.00		0.26		0.68		0.20		0.65	
MA				0.51		0.25		0.009		0.069		0.89	

[a,b]LSMeans within column and effect lacking a common superscript differ ($P \leq 0.05$).
[1]MA: Maternal age.
[2]MW: S = standard (breeder recommended) maternal prepubertal gain; H = maternal prepubertal BW gain 10% higher than the standard one.
[3]Sex: F = Female; M = Male.

the ones of younger mothers (35 wk). Female broilers from HMW hens had a lower FCR from 7 to 35 d than that of SMW broilers (1.437 vs 1.444 g:g for HMW and SMW broilers, respectively). This might have happened since female offspring from HMW hens had 0.69% lower ADFI over the course of 7 to 35 d and 5.8% lower abdominal fat deposition (Table 4) compared to their counterparts from the SMW hens; fat deposition in the body is energetically expensive, at approximately 9.2 kcal/g of BW gain, in contrast with lean tissue, which is composed of protein (4.1 kcal/g), and water (0 kcal/g) (Zuidhof et al., 2014). For every wk that MI was advanced, FCR of female broilers of HMW decreased by 0.0001 g:g but increased 0.021 g:g for males ($P = 0.059$). Notably, male chicks from HMW had greater gut weight compared to their SMW counterparts ($P = 0.048$, Table 4) which could potentially increase the overall FCR by increasing maintenance cost of the digestive tract.

Carcass Components

Broilers from HMW hens tended to have heavier breast muscles than that of from the SMW hens ($P = 0.071$, Table 4). A similar trend was observed on interaction of MI and sex on breast muscle weight ($P = 0.067$). For every week that the MI was advanced, breast muscle weight increased by 4.9 g for males, and decreased by 6.12 g for females. This is consistent with findings of van Emous et al. (2015) and Spratt and Leeson (1987) that male and female offspring responded differently to maternal nutrition, which may be related to epigenetic sex-specific genes that affect body composition in the offspring (van der Waaij et al., 2011). However, no effect was seen on proportional breast as a percent of live BW (Table 5).

The current data showed that increasing MW by 10% or advancing MI did not reduce abdominal fat content. This did not support our hypothesis that the offspring fat deposition would increase as a result of lower nutrition level (SMW) in breeders. van der Waaij et al. (2011) and Jing-feng et al. (2014) demonstrated that offspring of feed restricted breeders had relatively more abdominal fat deposition compared to those of breeders fed ad libitum due to a mismatch between maternal and offspring feeding level. It could be concluded that the maternal and offspring feeding level were not sufficiently different to reduce offspring abdominal fat pad weight in the current study. Since the goal of broiler production is to produce lean meat, an increase in broiler fat pad weight is not desired. Although SMW did not increase fat deposition in offspring broilers, based on the results of the current study, HMW still is recommended in broiler breeder industry due to its effect on reducing FCR in females.

Male broilers of HMW had a greater gastrointestinal tract (**GIT**) weight than those of SMW group, however, no effect was observed in females (Table 4). The gut is responsible for nutrient absorption, which plays a key role in metabolism to support growth and muscle development. A larger gut may have allowed the HMW hen offspring to make more efficient use of their feed due to the larger surface area of the gut, subsequently increasing their 35 d BW. Similarly, male broilers proportional GIT weight tended to increase by 0.08% of the live BW/wk of advanced MI ($P = 0.058$, Table 5).

Chicks from 42 wk old breeders had higher liver and heart weights than those of 35 wk old hens (Table 4). Proportional liver weight of broilers from 42 wk of age breeders was on average 1.05 times greater than that of broilers from 35 wk of age (Table 5). Advancing MI tended to increase male broilers of HMW liver weight compared to that of SMW ($P = 0.057$). For females and males, heart weight increased by 0.06 and 0.24 g/wk of advanced MI, respectively. The liver is an important metabolic organ that supports growth and development. A heavy BW broiler might have a larger liver to support greater maintenance requirements compared to a low BW broiler. Thus, greater liver weight might be related to greater BW on d 35 of male broilers from breeders whose MI was advanced.

The mechanism behind the effect of maternal environmental and nutritional conditions would either be through altered egg composition (O'Sullivan et al., 1991; Ekmay et al., 2013, 2014) or epigenetic mechanisms (Ferguson-Smith, 2011). Epigenetic effect can be passed onto the offspring. Epigenetic mechanisms are defined as alterations in the gene expression profile of a cell that are not caused by changes in DNA sequence; DNA methylation is an example of an epigenetic mechanism (Otterdijk and Michels, 2016; Pang et al., 2017). There was no effect of MW or MI on egg weight and egg components, differences in BW at hatch, 21, and 35 d of age suggest an epigenetic mechanism. Further research needs to clarify the underlying mechanisms of maternal effects of less feed restriction on offspring growth performance and carcass composition, with specific emphasis on epigenetics. The results of this study have a substantial implication for broiler enterprise in terms of productivity of the progeny chicks.

To investigate the effects of maternal growth patterns downstream in the broiler supply chain, the current experiment focused on relaxed maternal growth restriction during the prepubertal growth phase and earlier pubertal growth in breeders on their offspring growth and carcass traits. To our knowledge, this is the first investigation of the maternal effects of strategically designed growth trajectories based on advancing the timing of the pubertal growth phase in breeders. Overall, the current results indicate that increasing maternal prepubertal phase BW gain by 10% and advancing maternal pubertal phase inflection from 22 to 18 wk of age can increase male broiler growth rate and some carcass components weight in offspring chicks.

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DISCLOSURES

The authors declare that there is no conflict of interest.

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