# METABOLIC BASIS FOR DIFFERENCES IN FEED EFFICIENCY AND BODY COMPOSITION IN LEAN AND FAT BROILER CHICKENS PRODUCED BY DIVERGENT SELECTION FOR PLASMA VERY LOW DENSITY LIPOPROTEIN CONCENTRATION

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## SUMMARY

Divergent selection of broiler chickens for plasma very low density lipoprotein (VLDL) concentration has produced a 3 to 5-fold difference in rate of hepatic VLDL secretion and major changes in body composition and feed efficiency. Selection for low -VLDL concentration appears to have reached a limit because VLDL concentration in the plasma no longer reflects rates of VLDL secretion and further progress in reducing body fat content will probably require selection directly for low rates of VLDL secretion.

## INTRODUCTION

Excessive fatness is a problem in meat-type broiler chickens, as it is with other species of livestock. Modern commercial strains of broiler at commercial killing ages in the UK typically contain 130-200 g ether-extractable fat/ kg body weight and higher levels are often reported from the US. Much of this fat is derived from the diet or synthesised in the liver and is transported to the adipose tissue in the circulation in the form of triglyceride -rich portomicrons and very low density lipoproteins(VLDL). The latter predominate in the plasma of birds fed low fat diets.

In previous studies we have shown that the concentration of VLDL in the circulation of fully fed broiler chicken is sufficiently well correlated (r =0.6-0.7 ) with body fat content to act as an indirect measure of fatness in live birds (Griffin and Whitehead,1982). Subsequent selection of a commercial broiler male grandparent line for high or low plasma VLDL concentration for 10 generations has produced two lines with a greater than 10-fold difference in plasma VLDL concentration and major differences in body composition (Whitehead and Griffin,1984; Whitehead,1988). More importantly, the leaner,low-VLDL line has a significantly better efficiency of feed and protein conversion efficiency relative to the fatter, high-VLDL line (+14 and + 27% respectively). Current studies on the low- and high-VLDL lines are concentrating on determining the metabolic and hormonal mechanisms responsible. The present paper describes work comparing plasma lipoprotein metabolism in the two lines.

## MATERIALS AND METHODS

The birds used were from the 10th generation of selection and from the current M4 line from which they originated.Chicks were reared on a 23 h L:1h D photoperiod on the same low fat diet used during selection. Feed and water were available ad libitum and experiments were performed when the birds were 6-7 weeks of age (i.e. the same age that selection had been performed).

Rates of lipoprotein secretion were determined by measuring the rate of accumulation of triglyceride in the plasma after intravenous injection of sufficient anti-lipoprotein lipase antiserum to totally block VLDL clearance from the circulation. The effects of selection on VLDL metabolism were studied by determining the fate of intravenously-injected VLDL labelled in vivo with [14C]-palmitate. Hepatic lipogenesis was measured using [3H20]. Details are described by Griffin et al.(1989) and Asante and Griffin (1988).

## RESULTS AND DISCUSSION

The effect of divergent selection for plasma VLDL concentration on VLDL secretion rates, the fate of circulating VLDL and on body composition in males is shown in Table 1. The M4 line is not a true control, since it has been subject to selection by normal commercial criteria during the development of the two experimental lines. It now grows faster than the low- and high-VLDL lines, but this is unlikely to have significantly affected body composition or the mechanisms controlling body composition.

Table 1: Responses to selection for high- and low-plasma VLDL for 10 generations

| | Low-VLDL | M4 | High-VLDL |
|---|---|---|---|
| Body weight (kg) | $1.726^{A}$ | $2.133^{B}$ | $1.719^{A}$ |
| Body lipid (g/kg BW) | $124^{A}$ | $163^{B}$ | $229^{C}$ |
| Abdominal fat (g/kg BW) | $13.2^{A}$ | $16.3^{A}$ | $31.5^{B}$ |
| Plasma VLDL concentration ($\mu$moles TG/ml) | $0.18^{A}$ | $0.84^{B}$ | $4.19^{C}$ |
| VLDL secretion rate ($\mu$moles TG/h/ml of plasma) | $2.0^{A}$ | $3.8^{B}$ | $9.1^{C}$ |
| % of [14C]-VLDL | | | |
| -taken up by abdominal fat pad | $6.2^{A}$ | $5.7^{A}$ | $12.9^{B}$ |
| -oxidised to CO2 within 8 h | $15.0^{A}$ | - | $17.0^{A}$ |

Values are the means of 4-8 birds/line. Significant differences are indicated by different superscripts.

The rate of VLDL secretion in the high-VLDL line males is about 5-fold greater than in low-VLDL line males. Moreover, a much greater proportion of VLDL triglyceride is taken up into the abdominal fat pad in the high -VLDL line. This is not a consequence of a greater activity of lipoprotein lipase in these birds, since total depot activity is actually somewhat greater in the low-VLDL line (Griffin et al.,1989). Lipoprotein lipase activity in muscle is also significantly greater in the low-VLDL line. However, this does not appear to have increased the uptake and oxidation of VLDL triglyceride by muscle, since very similar proportions of [14C]-labelled VLDL were oxidised to [14CO2] in the low-and high-VLDL lines.

Comparisons of female broilers at the 10th generation of selection showed a

3-fold difference in VLDL secretion rate between high- and low-VLDL lines, suggesting that selection had been less effective in altering hepatic lipid metabolism than in males. The rate of hepatic lipogenesis in high-VLDL line females was only 2-fold greater than that in low-VLDL line females. Since dietary intake of fat is only slightly greater in the high-VLDL line (c.14%), differences in lipogenesis alone cannot account for between-line differences in rate of VLDL secretion.

$\beta$-hydroxybutyrate concentrations in avian plasma are much higher than those in mammalian plasma and this suggests that oxidation is a major fate of fatty acids in avian liver, even in the fully fed state. Plasma $\beta$-hydroxybutyrate concentrations in the low-VLDL line are also significantly greater than those in the high-VLDL line ( Griffin et al.,1989). These observations suggest that the lower rate of VLDL secretion in the low-VLDL line is due to both a reduction in rate of lipogenesis and the direction of an increased proportion of fatty acids available to the liver towards mitochondrial oxidation rather than lipoprotein synthesis. These changes are presumed to have a sparing effect on hepatic amino acid oxidation that in turn is responsible for the improved efficiency of amino acid utilisation for growth in the low-VLDL line.

Monitoring of the progress of the two lines during selection has shown that the increase in plasma VLDL and body fat content in the high-VLDL line has been achieved steadily over the 10 generations (Whitehead, 1988). In contrast, most of the progress in reducing plasma VLDL concentration and body fat content in the low-VLDL line was made in the first 3-4 generations. Comparison of plasma lipoprotein metabolism in the two lines suggests that selection for low-VLDL has reached a limit because plasma concentrations of VLDL no longer reflect rate of VLDL secretion( Griffin et al.,1989). Further progress in this line might be achieved by selection for low rate of lipoprotein secretion directly, using the anti-lipoprotein lipase antibody approach. However extrapolation of the data in Table.1 suggests that birds in which no lipoprotein was secreted into the plasma would still have about 100 g ether-extractable fat/kg of body weight. The most obvious source of this fat is adipose tissue lipogenesis. Most authors have concluded that this makes only a minor contribution to adipose tissue growth in birds, but this may not be the case in leaner strains. The prospects for further reduction of fatness by manipulating plasma lipoprotein metabolism may therefore be more dependent on the response of lipid metabolism in adipose tissue than of that in the liver.

## REFERENCES

ASANTE, E and GRIFFIN, H.D.1989. In Leanness in Domestic Birds (Leclercq,B. and Whitehead, C.C.,eds) pp.229-231, Butterworths, London.
GRIFFIN, H.D. and WHITEHEAD, C.C.1982. Br.Poultry Sci.23:307-313.
GRIFFIN, H.D., ACAMOVIC, F., GUO, K. and PEDDIE, J. 1989. J.Lipid Res.30: 1243-1250.
WHITEHEAD, C.C. and GRIFFIN, H.D. 1984. Br.Poultry Sci. 25:573-582.
WHITEHEAD, C.C..1988. In Leanness in Domestic Birds (Leclercq, B. and Whitehead, C.C.,eds) pp.175-202, Butterworths, London.