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Author:	S. Corbett, G.E. Gardner, C. Jose and D.W. Pethick
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High levels of dietary urea does not reduce muscle glycogen concentration

S. Corbett[A], G.E. Gardner, C.Jose and D.W. Pethick
Australian Cooperative Research Centre for Sheep Industry Innovation
School of Veterinary and Biomedical Science, Murdoch University, Murdoch, WA 6150
[A] Corresponding author. Email: scorbett@castledowns.com.au

Urea is commonly used in ruminant rations as a source of readily degradable nitrogen, However if it is not balanced correctly within the ration it can lead to high levels of ammonia production and absorption. In rats subclinical hyperammonaemia has been shown to reduce muscle glycogen as a result of reduced insulin responsiveness (Visek 1984). If this was to occur in sheep, the reduced muscle glycogen concentration could result in an elevated pH 24 hours post-slaughter (pH24) leading to a condition known as dark cutting, which negatively effects meat quality. Thus we hypothesise that sheep fed higher levels of urea will have higher plasma urea and ammonia concentrations, leading to reduced insulin response, reduced muscle glycogen stores at slaughter and higher pH24.

72 merino lambs were divided into 12 pens and fed one of 3 diets (10MJ/kg ME) containing 0, 3 or 4% dietary urea. Two lambs per pen were relocated to individual pens, and given 0.35g/kg dextrose intravenously on day 17. Blood was sampled 17 times between -30 and 130 minutes relative to administration of dextrose to determine glucose clearance rate, ammonia and urea concentration. On day 21 lambs were returned to their original group pens and then slaughtered commercially on day 33. Post-mortem muscle glycogen concentration and pH24 were measured in the *m. longissimus dorsi*. All measurements were analysed using a linear mixed effects model with diet as a fixed effect, sire, and dam as random terms, and plasma urea and ammonia used as covariates where applicable.

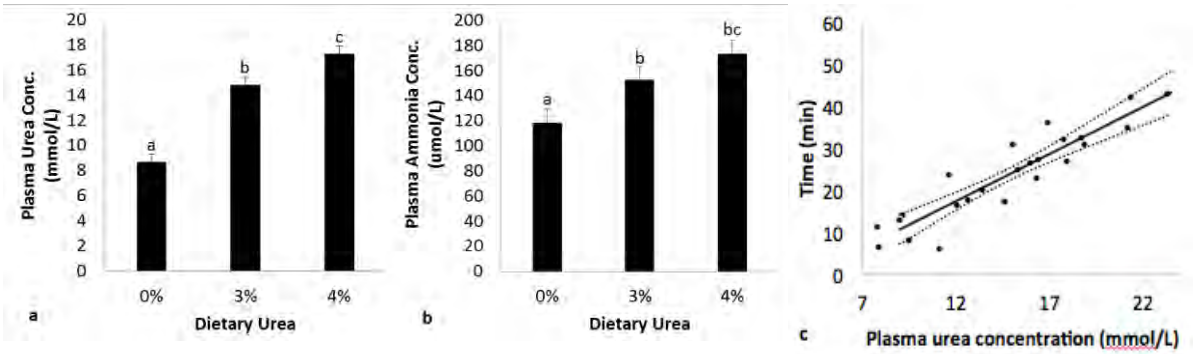

Figure 1 Effect of dietary urea on a) plasma urea concentration (mmol/L) and b) plasma ammonia concentration (umole/L). c) Effect of plasma urea (mmol/L) on glucose clearance time (min). Bars & dashed lines represent SE.

Plasma urea and ammonia increased by 98% ($P<0.05$) and 46% ($P<0.01$), when increasing dietary urea from 0 to 4% (Figure 1a and 1b). Across the plasma urea range glucose clearance time increased by 36 mins ($P<0.05$) (Figure 1c) indicating a decreased insulin response, an association not evident with ammonia. Dietary urea didn't affect muscle glycogen or pH24.

Contrary to our hypothesis muscle glycogen concentration and pH24 were not affected by dietary urea, in spite of increasing plasma urea reducing insulin response as demonstrated through increased glucose clearance time. The lack of response in muscle glycogen is in contrast to previous work in cattle (Gardner *et al*. 2013) and rats (Visek 1984). However, this may be explained by the low-stress handling of the lambs in this experiment depressing glycogen turn-over and limiting the potential expression of dietary urea treatment effects. Whilst further work is required to examine the effect of urea under commercial conditions, it is concluded that supplementing dietary urea under these "low-stress handling" conditions does not increase the risk of dark cutting.

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