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ORIGINAL RESEARCH

Swelling disease of Angora goats is characterised by panhypoproteinaemia, leukocytosis, and hyperaldosteronaemia

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Background: Swelling disease is a poorly understood clinical syndrome of South African Angora goats that is characterised by acute-onset diffuse oedema. Adrenocorticotropic hormone-stimulated aldosterone secretion has been proposed as a possible aetiology of swelling disease, but this hypothesis has not been tested.

Objectives: To investigate aldosterone secretion in swelling disease by reanalysing data collected during a 2015 clinical investigation of the syndrome in which serum aldosterone and cortisol levels were measured along with standard clinical laboratory variables in affected and unaffected animals.

Methods: Goats affected with swelling disease, and unaffected herd-mates, were sampled for measurement of haematology variables, serum protein variables, and serum sodium, aldosterone, and cortisol. Results for affected and unaffected goats were compared using the Mann-Whitney U test, and the association between variables was investigated using Spearman's rank-order correlation analysis.

Results: Goats affected with swelling disease had significantly higher white blood cell counts (WBC) and serum aldosterone than unaffected animals, and significantly lower total serum protein (TSP), albumin, globulin, and albumin/globulin ratios. For all animals (n = 60), serum protein variables showed a significant and strong positive correlation with one another, and a negative correlation with serum aldosterone and WBC.

Conclusion: Swelling disease of South African Angora goats is characterised by panhypoproteinaemia, a low A/G ratio, leukocytosis, and hyperaldosteronaemia. Causal relationships between variables could not be determined, however, the detection of hyperaldosteronaemia provides novel insight into the possible pathophysiological mechanisms of this syndrome.

Keywords: aldosterone, Angora goat, oedema, swelling disease

Introduction

Swelling disease is a clinical syndrome of South African Angora goats that is characterised by the acute onset of oedema, often affecting numerous individuals in a herd, and typically associated with stressful events such as shearing, weaning, and inclement weather (Snyman 2004; Snyman & Snyman 2005). Clinical and laboratory findings vary in severity and include subcutaneous oedema, ascites, hypoproteinaemia, neutrophilia, and, in severe cases, death (Mitchell et al. 1983; Snyman 2004; Vermeulen 1986). Post-mortem examinations commonly confirm these findings, revealing moderate to severe subcutaneous and mesenteric oedema along with ascites (Snyman 2004). While verminosis, coccidiosis, and diet have been investigated as possible causes of the syndrome, no definitive aetiology or pathophysiological mechanism has been elucidated (Snyman & Snyman 2005).

Given that the syndrome is associated with stressful events, adrenocorticotropic hormone (ACTH) release has been proposed as the trigger for swelling disease, and hypothesised to result in elevated aldosterone secretion, sodium and water retention, hypertension, and oedema formation (Thompson 1994). To test this hypothesis, the present study reanalyses data collected during a 2015 clinical investigation in which serum aldosterone and cortisol levels, along with standard clinical laboratory variables, were measured in animals both affected and unaffected by swelling disease.

Materials and methods

Animals originated from two farms in the Sarah-Baartman District of the Eastern Cape Province, South Africa. Herd 1 (Farm A) comprised weaned kids, and Herds 2 and 3 (Farm B) comprised weaned kids and yearling does, respectively. The occurrence of swelling disease, i.e. diffuse subcutaneous oedema in numerous individuals was reported by the owners in May and June of 2015, and samples were collected from Herds 1 and 2 within five days, and from Herd 3 within one day of the onset of clinical signs. Blood was collected by jugular venipuncture from visibly affected (n = 10) and visibly unaffected animals (n = 10) from each herd, into serum tubes and blood collection tubes containing ethylenediaminetetraacetic acid (EDTA).

Samples were analysed at IDEXX laboratories, Johannesburg, South Africa, within 48 h of collection. Complete blood counts were performed using an XT 2000iV automated haematology analyser (Sysmex, Kobe, Japan) and serum sodium, total serum protein (TSP), and serum albumin were measured using a Vitros

350 dry slide analyser (QuidelOrtho, San Diego, USA). Serum globulin concentration was calculated by subtracting the albumin value from the TSP. Serum cortisol and aldosterone were measured using the Immulite 1000 Cortisol immunoassay (Siemens, Munich, Germany) and Liaison Aldosterone assay (DiaSorin, Saluggia, Italy), respectively.

Results were interpreted with reference to normal values previously reported for goats (Alberghina et al. 2010; Jackson & Cockcroft 2002a; Jackson & Cockcroft 2002b). For each herd, blood variables from affected and unaffected animals were compared using the Mann–Whitney U test (GraphPad Prism version 10.4.1 for Windows, GraphPad Software, Boston, Massachusetts USA, www.graphpad.com). For all animals (n = 60), the association between variables was investigated using Spearman's rank-order correlation analysis (GraphPad Prism). A p-value of < 0.05 was regarded as statistically significant.

Results

Of the 60 laboratory reports received, 44 described mild to severe haemolysis, although it was not stated whether this referred to the serum or EDTA sample. Apparent outliers were not removed from the data set and all data used for further analysis are publicly available at https://doi.org/10.25403/UPresearchdata.29924324.v1.

For all animals, the median red blood cell count (RBC), packed cell volume (PCV), and mean cell haemoglobin concentration (MCHC) were 2.5 x 10^{12}/L, 8.3%, and 129 g/dL respectively, far lower (RBC and PCV) and higher (MCHC) than normal values previously reported for goats, and highly suggestive of marked

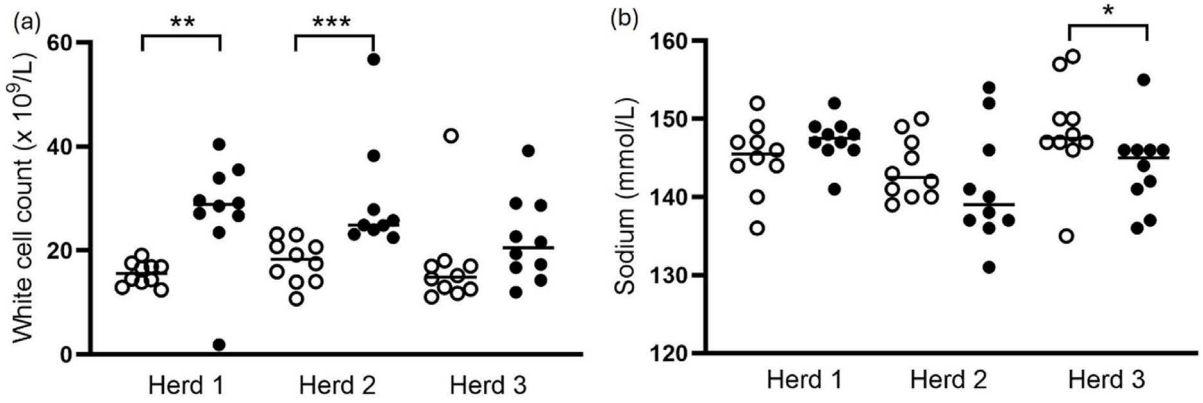

Figure 1: White blood cell counts (a) and serum sodium (b) for Angora goats from three herds, that were either unaffected (o), or affected (•), by swelling disease. Median values are shown. *, $p < 0.05$, **, $p < 0.01$, ***, $p < 0.001$.

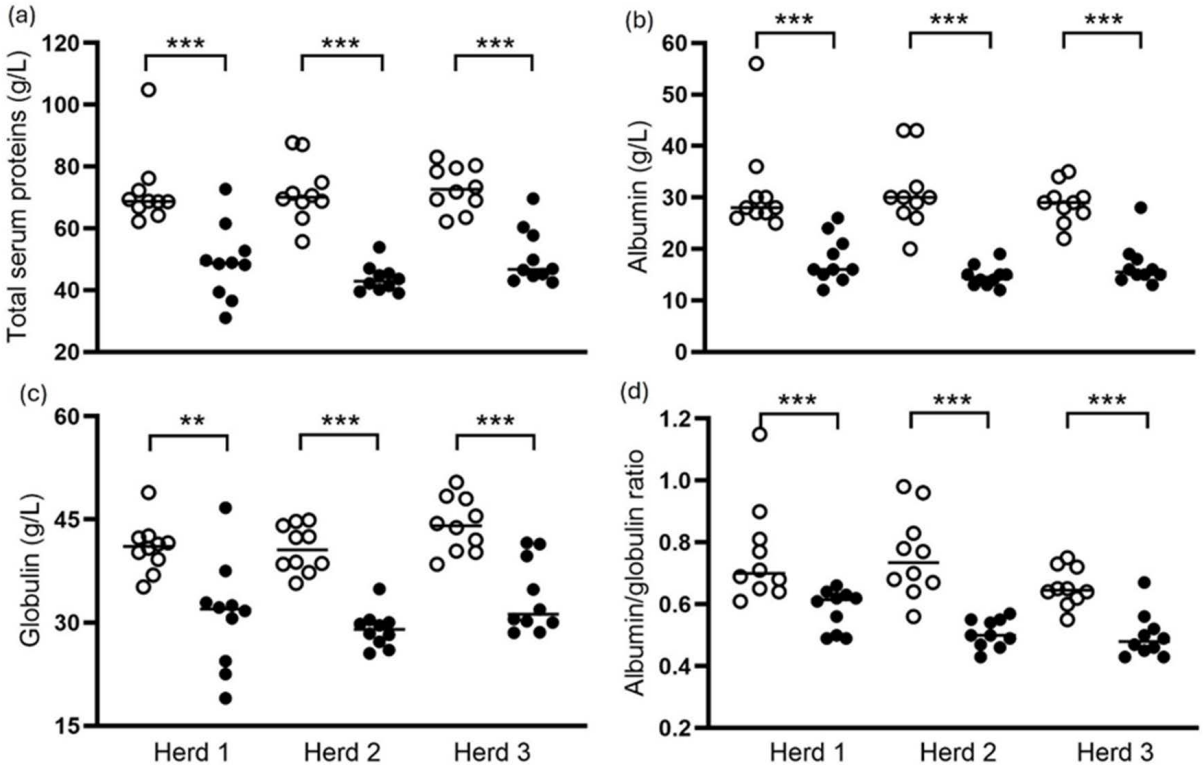

Figure 2: Total serum protein (a), serum albumin (b), globulin (c), and albumin/globulin ratios (d) for Angora goats from three herds, that were either unaffected (o), or affected (•), by swelling disease. Median values are shown. **, $p < 0.01$, ***, $p < 0.001$.

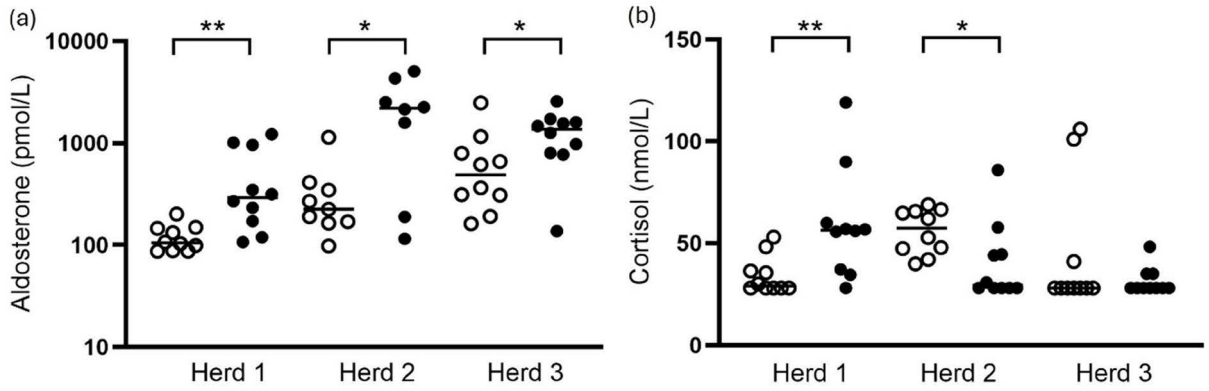

Figure 3: Serum aldosterone (a) and cortisol (b) for Angora goats from three herds, that were either unaffected (o), or affected (•), by swelling disease. Median values are shown. *, $p < 0.05$, **, $p < 0.01$.

Table I: Spearman's ranked correlation coefficient (r) for TSP (g/L), albumin (g/L), globulin (g/L), A/G ratio, sodium (mmol/L), HGB (g/L), WBC (x 10^9/L), cortisol (nmol/L), and aldosterone (pmol/L) from Angora goats both affected ($n = 30$) and unaffected ($n = 30$) by swelling disease. *, $p < 0.05$, **, $p < 0.01$, ***, $p < 0.001$.

	TSP	Albumin	Globulin	A/G ratio	Sodium	HGB	WBC	Cortisol
Albumin	0.95***							
Globulin	0.96***	0.87***						
A/G ratio	0.72***	0.85***	0.56***					
Sodium	0.15	0.15	0.19	0.11				
HGB	-0.10	-0.02	-0.17	0.10	-0.01			
WBC	-0.57***	-0.60***	-0.53***	-0.50***	-0.30*	0.04		
Cortisol	-0.03	0.02	-0.10	0.22	-0.15	0.06	0.38**	
Aldosterone	-0.53***	-0.57***	-0.47***	-0.58***	-0.39**	0.21	0.44***	0.01

in vitro haemolysis (Jackson & Cockcroft 2002a). The RBC, PCV, mean cell volumes, and MCHC were therefore regarded as spurious and excluded from the study. All animals displayed a normal haemoglobin concentration (Jackson & Cockcroft 2002a, Supplementary Table) with no significant difference in these values between affected and unaffected individuals in each herd ($p \geq 0.15$ for all herds). For each herd, median WBC of unaffected individuals were greater than normal values reported for goats (Jackson & Cockcroft 2002a, Supplementary Table). Goats affected by swelling disease had significantly higher white blood cell counts (WBC) than unaffected animals (Figure 1a).

All serum sodium concentrations ($n = 60$) were within the normal range reported for goats (Jackson & Cockcroft 2002b, Supplementary Table), although affected yearling does from Herd 3 displayed significantly lower values than unaffected animals (Figure 1b). Unaffected animals from all herds had TSP, albumin, globulin, and albumin/globulin (A/G) ratio values consistent with normal values for goats (Alberghina et al. 2010, Supplementary Table), however, within each herd, these were significantly lower in affected animals (Figure 2).

Serum aldosterone concentrations were significantly greater in affected animals in all three Herds (Figure 3a). Cortisol results ranged from 13.9 to 106 nmol/L, however, in many instances, cortisol was reported as < 27.6 nmol/L, and all results < 28 nmol/L were therefore corrected to 28 nmol/L for further analysis. Compared to unaffected animals, affected goats displayed significantly higher serum cortisol values in Herd 1, significantly lower values in Herd 2, and no difference in Herd 3 (Figure 3b).

Spearman's rank-order correlation analysis results are reported in Table I. For all animals ($n = 60$), TSP, albumin, globulin, and A/G ratio values showed a significant and strong positive correlation with one other, and a negative correlation with aldosterone concentration and WBC. Aldosterone and WBC were positively correlated and both variables were negatively correlated with serum sodium concentration. Serum cortisol showed a positive correlation with WBC.

Discussion

Compared to unaffected individuals, Angora goats with swelling disease displayed panhypoproteinaemia, low A/G ratios, elevated WBC, and hyperaldosteronaemia. Many of these findings are consistent with previous descriptions of such cases (Snyman 2004; Vermeulen 1986), while the detection of hyperaldosteronaemia provides a novel insight into the possible pathophysiology of this syndrome.

Affected animals displayed a significant reduction in TSP, with highly correlated serum albumin and globulin values. An absolute decrease in total protein could be indicative of a wide range of conditions, including reduced protein synthesis, blood loss, and protein-losing nephropathies or enteropathies (Friedrichs et al. 2025). However, given the normal HGB levels, the spontaneous resolution of most swelling disease cases, and

the minimal organ pathology reported on necropsy (Snyman 2004), these causes appear unlikely.

Alternatively, serum protein dilution, and a relative reduction in both serum albumin and globulin values, as observed in the present cases, can result from plasma volume expansion (Friedrichs et al. 2025). In such a scenario, hypervolaemia might also contribute to oedema formation both directly as a result of increased hydrostatic pressure (Friedrichs et al. 2025; Starling 1896), and indirectly by triggering atrial natriuretic peptide release, which potentiates shedding of the endothelial glycocalyx and increased vascular permeability (Chappell et al. 2014).

Complicating the picture, despite albumin and globulin values being highly correlated, affected animals showed a significantly decreased A/G ratio. This disproportionate drop may indicate an absolute reduction in albumin, an absolute increase in globulins, or both. The current data do not allow for distinction between these possibilities, so various scenarios are considered here in relation to the possible pathophysiology of oedema formation in this condition.

Albumin contributes ~ 80% of the colloid osmotic pressure (COP) of plasma (Levitt & Levitt 2016) and has been described as the principal force opposing capillary hydrostatic pressure to maintain fluid balance across blood vessel walls (Starling 1896). As such, the reduction in COP associated with hypoalbuminaemia has been regarded as an important driver of oedema formation (Friedrichs et al. 2025; Steyl & van Zyl-Smit 2009). Indeed, in ruminants, hypoproteinaemia associated with gastrointestinal verminosis, largely due to chronic blood and protein loss into the gastrointestinal tract, is widely reported as the primary cause of submandibular oedema (Flay et al. 2022). However, there is limited empirical evidence for these assertions. Moreover, while oedema of nephrotic syndrome has traditionally been attributed to hypoproteinaemia, recent evidence highlights primary renal sodium and water retention as the central driver of its pathophysiology (Doucet et al. 2007; Bobkova et al. 2016), and dogs with moderate experimental hypoproteinaemia (circa 46 g/l) and a significantly reduced COP displayed no increase in interstitial fluid volume compared to controls (Manning & Guyton 1983). Similarly, analbuminaemic human patients show little or no oedema (Doucet et al. 2007; Levitt & Levitt 2016). Taken together, the lack of evidence for a causal link between hypoalbuminaemia and oedema indicates that despite their co-occurrence in this syndrome, oedema cannot confidently be interpreted as evidence of an absolute reduction in serum proteins, as has previously been suggested (Mitchell et al. 1983).

An alternative interpretation of the reduced A/G ratio is that it reflects a pro-inflammatory state driven by an absolute increase in globulins, with or without a concomitant reduction in albumin. Systemic and vascular inflammation can increase endothelial permeability, resulting in the redistribution of fluid and albumin into the interstitial space (Levitt & Levitt 2016; Soeters et al. 2019; Steyl & van Zyl-Smit 2009). This mechanism could contribute to an absolute hypoalbuminaemia through two pathways: first, by shifting albumin out of the vascular compartment, and second, through the suppression of hepatic albumin synthesis, reflecting its role as a negative acute phase protein in inflammatory states (Friedrichs et al. 2025).

Notably, Angora goats affected by swelling disease displayed a marked elevation in serum aldosterone, which might be explained as either a cause or consequence of the syndrome. In humans, capillary leak syndrome, characterised by increased capillary permeability with the shift of protein and fluid into the interstitium, results in hypoalbuminaemia, haemoconcentration, widespread oedema, and hypotension, triggering secondary aldosterone release via the renin-angiotensin-aldosterone system (RAAS) (Siddall et al. 2017). However, this syndrome is associated with extremely low serum protein levels (2 to 30 g/l) (Correia et al. 2023; Druey et al. 2016), and in comparison, dogs with moderate experimental hypoproteinaemia (46 g/l) maintained plasma volume and blood pressure independently of RAAS activation (Manning & Guyton 1983). The current absence of evidence of hypotension and RAAS activation during swelling disease precludes a definitive conclusion about this possibility.

Alternatively, a primary elevation of mineralocorticoids, including aldosterone, as seen in cytochrome P450 17 α-hydroxylase/17,20-lyase (CYP17) deficiency in humans, could cause sodium and water retention, plasma volume expansion, and hypertension (Kim & Rhee 2015). This enzyme regulates the differential biosynthesis of aldosterone, cortisol, and androgens in the adrenal cortex, and a functional deficiency can result in inadequate cortisol and androgen production and excessive aldosterone secretion (Kim & Rhee 2015; Storbeck et al. 2008). In addition to hypertension, hyperaldosteronaemia is associated with other mechanisms of oedema formation, including damage to the endothelial glycocalyx and increased endothelial permeability to albumin (Aleksiejczuk et al. 2022; Butler et al. 2019; Crompton et al. 2023). Indeed, increased capillary permeability has previously been proposed as a pathophysiological mechanism of swelling disease (Mitchell et al. 1983). Notably, the *CYP17* gene is duplicated in goats, and deleterious *CYP17* mutations have been described in South African Angora goats, with distinct genotypes differing in their ability to produce cortisol in response to hypoglycaemia (Storbeck et al. 2008). This variability might explain the differences in cortisol responses observed in the present study. The consequence of this deficit for aldosterone production has not been investigated in Angora goats but provides a plausible mechanism for the development of primary hyperaldosteronaemia in this breed.

Hyperaldosteronaemia also provides possible explanations for additional findings in this syndrome. Stressful events, such as shearing, resulting in increased ACTH release, would stimulate adrenal steroid biosynthesis, and could exacerbate primary hyperaldosteronaemia. In turn, aldosterone, and its precursors such as corticosterone, would activate both mineralocorticoid and glucocorticoid receptors of inflammatory cells, resulting in leukocytosis (Bene et al. 2014; Ince et al. 2019). Notably, this may explain the marked leukocytosis previously observed in apparently health South African Angora goats (Haw 2013; Parsons et al. 2023). Moreover, hyperaldosteronaemia is associated with oxidative-like stress of erythrocytes, possibly contributing to the high levels of haemolysis observed in the blood samples (Bordin

et al. 2013). In turn, given that aldosterone promotes sodium resorption, this might explain the counterintuitive negative correlation between aldosterone and serum sodium, as red cell lysis results both in dilution of serum sodium and measurement interference (Dimeski 2008; Kalaria et al. 2021). Unfortunately, in the present study, the absence of sensible data on intracellular haemoglobin precludes an objective analysis of haemolysis and its possible consequences.

In conclusion, this descriptive clinical study is limited by the challenges of remote sample collection and the lack of access to specialised veterinary laboratories in rural South Africa. While the study does not allow for definitive conclusions regarding causal relationships between variables, the detection of hyperaldosteronaemia in affected goats provides novel insight into the possible pathophysiology of swelling disease. Future studies should aim to better characterise the changes in serum protein concentrations observed in this syndrome, elucidate the underlying mechanisms of oedema formation, and investigate a possible association between hyperaldosteronaemia and the genetic CYP17 deficiency of South African Angora goats.

Conflict of interest

The authors declare no conflict of interest.

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Ethical approval

Prior to the commencement of the study, ethical approval was obtained from the Animal Ethics Committee of the University of Pretoria (REC 022-24).

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Supplementary Table:

Selected variables (median and range) for Angora goat kids (Herds 1 and 2) and yearling does (Herd 3), either affected, or unaffected by swelling disease, and associated normal reference values

	Herd 1		Herd 2		Herd 3		Reference
	Unaffected	Affected	Unaffected	Affected	Unaffected	Affected	values
HGB (g/L)	104 (11 - 112)	97 (40 - 112)	107 (78 - 115)	98 (63 - 114)	103 (86 - 142)	108 (89 - 124)	80 - 120[a]
WBC (x10^9/L)	15.6 (12.5 - 19.1)	28.9 (1.9 - 40.4)	18.3 (10.7 - 23.2)	24.9 (22.5 - 56.8)	14.9 (11.1 - 42.1)	20.5 (12.0 - 39.2)	4 - 13[a]
Sodium (mmol/l)	146 (136 - 152)	148 (141 - 152)	143 (139 - 150)	139 (131 - 154)	148 (135 - 158)	145 (136 - 155)	135 - 156[b]
TSP (g/L)	68.7 (62.2 - 104.9)	48.7 (31.0 - 72.7)	70.1 (55.7 - 87.7)	42.9 (39.0 - 53.9)	72.6 (62.2 - 83.0)	46.7 (42.5 - 69.6)	55 - 87[c]
Albumin (g/L)	28 (25 - 56)	16 (12 - 26)	30 (20 - 43)	15 (12 -19)	29 (22 - 35)	16 (13 - 28)	24 - 42[c]
Globulin (g/L)	41 (35 - 49)	32 (19 - 47)	41 (36 - 45)	29 (26 - 35)	44 (29 - 50)	31 (29 - 42)	none
A/G ratio	0.70 (0.61 - 1.15)	0.62 (0.49 - 0.66)	0.74 (0.56 - 0.98)	0.50 (0.43 - 0.57)	0.65 (0.55 - 0.75)	0.48 (0.43 - 0.67)	0.47 - 1.63[c]

[a]Jackson & Cockcroft 2002a, [b]Jackson & Cockcroft 2002b, [c]Alberghina et al. 2010.