Infectious Disease in Koalas: Implications for Conservation

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Abstract

Infectious disease in koalas is undoubtedly one of the critical threatening processes contributing to their dramatic population declines in Queensland and New South Wales. Two of the most important infections: koala retrovirus (KoRV) and *Chlamydia* are still relatively poorly understood, although they are the subject of active research at a number of universities. We still have much to learn about their associated disease pathogenesis, the interaction between these agents, their ecological impact and distribution. This information is important not only to assist in our efforts to treat affected koalas, but also to add weight to our arguments for greater protection of habitat. Our concerns about the impact of infectious disease in koalas continue to be validated by both koala admissions to the Australian Wildlife Hospital, and also our investigations of koala health in a number of wild koala populations. We will present an overview of the prevalence of disease in some populations that we are studying in south-east Queensland (SEQ) and the implications for koala conservation generally.

Introduction

Although the loss of koala habitat and consequent decline of the species has been a concern of conservationists for many years (Melzer et al., 2000), the actual magnitude of the decline and severity of the situation is just starting to be accepted by some of the regulatory authorities. Similarly, the high prevalence of disease in koalas has been recognised for well over a century, but its importance as a key threatening process has only recently received acknowledgement. Despite this, regulators and legislation at all levels of government continue to fail to address koala declines in any meaningful way. Although the principles and intent of ecologically sustainable development (ESD) are espoused commonly in government policy and statute, their application (in terms of effective and measurable outcomes) is virtually non-existent. As a consequence, the localised extinction of koalas is commonplace, and the tipping points for wider, or regional, extinction seem to be looming.

There is no question that the protection of remaining koala habitat and restoration of effective corridors between habitat remnants are the most critical things that must be done to conserve

koalas. In focusing on the prevalence and impact of disease in this paper, our intent is not in any way to detract from the importance of habitat issues. However, if the critical threat that disease plays in population decline and extinction is not considered in population modeling, conservation planning and prioritisation of research funding, it will be a recipe for disaster. We will focus our discussion on the two most important infectious agents in koalas: koala retrovirus and *Chlamydia*, because arguably they are having the greatest impact on wild koala populations.

Koala Retrovirus (KoRV)

Retroviruses are fragile organisms that are able to integrate their own genetic sequences into the DNA strands of the cell that they have infected. In doing so, they are able to hijack host cell processes to produce many more virus particles; in effect, turning the host cell into a virus factory. The genomes of retroviruses contain regions that strongly promote the transcription of the viral DNA sequence by the host cell. Whilst this process is designed to promote the production of virus particles, it may also "accidentally" switch on genes of the host cell, and this in turn may cause cancer. Conversely, the viral DNA may disrupt a host cell gene, leading to the death of the cell, or altered cell function. Simplistically, the clinical syndromes that are observed in koalas reflect these basic molecular processes.

The following conditions in koalas are thought to be caused by infection with KoRV:

- 1. Leukaemia (a cancer of the blood forming cells)
- 2. Myelodysplasia (abnormalities in production of blood cells)
- 3. Immunodeficiency syndrome (koala "AIDS")
- 4. Other cancers, including lymphoma, osteochondroma, mesothelioma.

A range of other conditions may be associated with KoRV infection in koalas, but it is beyond the scope of this paper to list them all. The relationship between KoRV infection and chlamydial disease is discussed below.

Epidemiology of KoRV

Infection with KoRV appears to be close to 100% in Queensland and NSW koalas, and somewhat less than that in Victorian and South Australian populations, based on recent work by the KoRV Research Group at the University of Queensland. Interestingly, the incidence of the conditions listed above is essentially zero in Victorian koalas (Bodley, K. 2009 pers. comm., 12 May), based on approximately 1500 wild koala examinations (throughout Victoria) each year.

All koalas tested by Rachel Tarlinton during her PhD studies were viraemic with KoRV (virus particles present in the blood), but the level of viraemia varied considerably between koalas (Tarlinton 2006). She also demonstrated that there was a significant correlation between a high level of viraemia and development of neoplastic diseases such as leukaemia (Tarlinton *et al.*, 2005).

Koala retrovirus has endogenised in koalas in Queensland and New South Wales. That is: it has infected germ line cells (spermatozoa or oocytes) and is transmitted genetically (by inheritance) from parents to offspring. Although this is a known mechanism of transmission, KoRV may also spread from koala to koala (horizontal spread) by close contact, and from infected mothers to their joeys via the milk, similarly to the way that many other viruses spread (Hanger 1999). Whether KoRV can be transmitted by biting insects has yet to be determined.

Where it came from, when it arrived, and how its impact will play out in the koala population, we still do not know.

KoRV and Chlamydiosis

It seems a reasonable hypothesis that *severe* chlamydial disease is more common in koalas because of the consequences of KoRV infection, specifically its effects on immune responses, and that normal immune function (in koalas without AIDS) results in more minor chlamydial pathology (similar to that seen in chlamydiosis in other species). This contention is supported by the generally minor nature of chlamydial pathology in southern koalas (Bodley, K. 2009 pers. comm. 12 May), where KoRV is less prevalent. Although Tarlinton's work suggested an association between chlamydial disease and high KoRV viraemia, the statistical significance of that association has not yet been demonstrated. Current research, which is using a larger and more sophisticated data set, may clarify the situation.

Chlamydia

Chlamydial infection in koalas is common and affects most mainland and many island koala populations. Prevalence of *infection* and prevalence of *disease* varies between populations, but severe disease is more common in northern koalas (Qld and NSW) than in southern koalas (Vic and SA), irrespective of prevalence of infection (Timms 2000). Severe cystitis, keratoconjunctivitis and active reproductive tract disease is common in northern koalas, and very uncommon in southern koalas (Bodley, K. & Lynch, M. 2009 pers. comm. 12 May). Although cystic change and fibrosis of the reproductive tract leading to infertility is common in southern koalas, the severe debilitating pathology seen commonly in northern koalas is not.

Chlamydial disease in southern koalas is more consistent (in terms of severity of pathology) with chlamydial disease in humans (*Chlamydia trachomatis*) and other species (Timms, P. 2009,

pers. comm. 12 May). That is: it is of a relatively minor nature and rarely causes debilitating disease. In contrast: severe chlamydial disease seen commonly in northern koalas is quite unusual, compared with other species. ^a

Chlamydial infection in koalas is commonly associated with ocular infections (keratoconjunctivitis), urinary tract infection (cystitis and nephritis) and reproductive tract disease (prostatitis, metritis, pyometron, cyst formation and fibrosis). Infertility is a common sequel to reproductive tract infection in both northern and southern koalas. Less common manifestations include respiratory infections and granulomas, and the epizootic koala "flu" is thought to result from acute infection, possibly with a strain of *Chlamydia pneumoniae* (Nicholson, V. 2009, pers. comm. 1 May).

There are many other papers and articles that describe in detail chlamydial epidemiology, pathology, and treatment in koalas. It is beyond the scope of this paper to provide such detail. The most important points regarding chlamydial disease in koalas are:

- 1. It is common and widespread in most koala populations.
- 2. Severe debilitating disease is more common in koalas in Qld and NSW than in southern states.
- 3. KoRV co-infection probably increases the risk of serious disease.

Prevalence of infection versus prevalence and incidence of disease

Koalas can be infected with *Chlamydia* and also KoRV without detectable disease, hence prevalence of *disease* is less than prevalence of *infection*. Many studies have reported *Chlamydia* infection prevalence in wild koala populations using a variety of detection methods (Devereaux *et al.*, 2003; Timms 2000; White & Timms 1994), and some have also described the prevalence of overt disease (Jackson *et al.*, 1999). However, with the exception of a study by Jones (2008), no wild koala population health surveys, to date, have routinely used techniques such as ultrasound and cystocentesis to detect pathology which might otherwise be inapparent. These techniques are essential in the routine assessment of koalas for urogenital tract disease. Hence, many of the aforementioned studies are likely to have underestimated disease prevalence due to insensitivity of detection methods. Furthermore, some koalas, which, at the time of examination show no signs of disease, but are *Chlamydia* positive, may progress to clinically diseased in time.

^a This generalisation does not apply to disease resulting from cross-species transmission of some types of *Chlamydia*, such as psittacosis in humans, which is caused by infection with avian strains of *Chlamydia psittaci*, and can cause severe and occasionally fatal infections.

Prevalence of Disease in Two SEQ Koala Populations

One of our current research projects is investigating the prevalence and incidence of disease in two koala populations in the Moreton Bay Regional LGA, SEQ, one in the suburb of Brendale, the other in Narangba (hereafter referred to as Populations A and B respectively). In an attempt to capture all of the koalas in each population, comprehensive searches of both sites were undertaken. After capture, each koala was transported to the Australian Wildlife Hospital and subjected to a thorough clinical assessment using a standardised veterinary protocol (Appendix 1). This included a complete physical examination under general anaesthesia, and a range of ancillary diagnostic tests designed to detect most known conditions in koalas. Clinical data were recorded for 25 koalas in Population A (13 male, 12 female) and 17 koalas in Population B (7 male, 10 female).

A high prevalence of chlamydiosis was found in both populations: 44% (5 male, 6 female) and 41% (2 male, 5 female) of koalas were found to have chlamydial disease in Population A and B, respectively (Table 1). Despite the high level of chlamydial disease in both populations, of the koalas with detectable illness, 45% (1 male, 4 female) of Population A, and 57% (1 male, 3 female) of Population B exhibited no *overt* signs of disease (Table 2). These results indicate that without thorough investigative veterinary techniques, subclinical disease would in some cases have remained undetected.

	Population A	Population B
Total No. of Koalas	25 (13 male: 12 female)	17 (7 male: 10 female ^b)
Healthy Koalas (no detectable disease)	52% (7 male: 6 female)	59% (5 male: 5 female)
Diseased Koalas Requiring Veterinary Intervention	48% (6 male: 6 female)	41% (2 male: 5 female)
Chlamydial Disease	44% (5 male: 6 female)	41% (2 male: 5 female)
No. of Females with a Joey	33% (4 females)	40% (4 females)
Euthanased/Died due to Severity of Disease	32% (3 male: 5 female)	11% (0 male: 2 female)

Table 1: Koala Health Summary and Outcomes of Population A and Population B

^b This number includes one new case of disease detected at the second (6 month) health check

Of the female koalas, 50% (6/12) from Population A, and 50% (5/10) from Population B had reproductive tract disease. Of these, only 33% (2/6) of Population A and 40% (2/5) of Population B demonstrated overt physical signs of chlamydial disease, and those signs were referable to cystitis (the koalas had "dirty-tail")(Table 2). The remainder of cases required palpation and/or ultrasonography to make the diagnosis. In other words, overt signs of reproductive tract disease are rare, and sometimes cystitis is also not apparent as dirty-tail.

Chlamydial Disease	Population A		Population B	
	Males	Females	Males	Females
Overt Chlamydial Disease	4/5 (80%)	2/6 (33%)	1/2 (50%)	2/5 (40%)
Subclinical Disease	1/5 (20%)	4/6 (66%)	1/2 (50%)	3/5 (60%)
Conjunctivitis only	1/5 (20%)	0/6 (0%)	1/2 (50%)	0/5 (0%)
Cystitis only	4/5 (80%)	0/6 (0%)	1/2 (50%)	0/5 (0%)
Multifocal Chlamydial Disease	0/5 (0%)	4/6 (66%)	0/2 (0%)	2/5 (40%)
Reproductive Tract Disease (likely to be infertile)	0/5 (0%)	6/6 (100%)	0/2 (0%)	5/5 (100%)
Total Koalas with	5	6	2	5°
Chlamydiosis	TOTAL (POP A)= 44%	TOTAL (POP B)	=41.2%
	11 out of 2 population A wit	5 individuals in h chlamydiosis	7 out of 17 individ B with chlamydios	luals in population

Table 2: Summary of Koalas with Chlamydial Disease in Population A and Population B

Finally, the definitive indicator of fertility (production of a joey) was shown by only 33% (4) and 40% (4 - one of which subsequently became infertile) of females of breeding age in populations A and B respectively. In population A, one female was not yet of breeding age, and one had a lesion on ultrasound which could not, at the time, be distinguished from a pregnancy. That female has not since produced a joey, so, in retrospect, the lesion is likely to have been a pathological change. In population B, two females were not of breeding age.

^c This number includes one new case of disease detected at the second (6 month) health check.

Our disease results contrast starkly with those of Lane (2008), who performed population surveys and disease prevalence estimates in the Pine Rivers, Caboolture and Redcliffe Shires in 2007 (in which our study populations are located). The primary tool for detection of disease presence in that study was a pair of binoculars, and, predictably, their estimate of disease prevalence was low (approximately 10% of "urban" and "bushland" koalas combined). The fact that they reported low observed disease prevalence, without qualifying that their sensitivity for detection of disease was very limited, means that *true* disease prevalence may be (and has been) misinterpreted as also being low.

For this reason prevalence studies that rely on the observation of overt signs of chlamydial disease will invariably underestimate disease prevalence in the sample group. Koalas that have subclinical reproductive tract pathology are clearly of significance to disease prevalence studies, and to the assessment of population health. In most cases, these females will be infertile, population fecundity will be affected proportionately with prevalence, and therefore impacts on population viability can be significant, as demonstrated by our data.

Incidence of disease

With disease in wild populations it is often useful to investigate the *incidence* of disease, that is: the number of new cases per population over a given time period. For example: in our koala disease study described above, the incidence of new cases of infertility in female koalas was 10% per year (one new case among the 10 female koalas during the year). For chlamydiosis, a chronic disease, data about both *incidence* (of new cases) and *prevalence* (of present disease) are useful: the first give us an estimate of what might happen to a population over time, the latter, a snapshot or cross-sectional view of the population at a given moment in time. In contrast, the *incidence* of some acute KoRV-associated diseases (such as leukaemia) is more important than the *prevalence*, because they kill koalas relatively quickly, therefore the chance of detecting affected animals in a cross-sectional study is small. With KoRV-associated AIDS, prevalence data is just as important, because it (probably) takes longer to kill koalas, and information on how many koalas in a population have AIDS at any given point in time, is useful.

If we are to be well-informed about the impacts of disease on koala populations and the likely consequences for survival (or extinction), then we need to gather data on both incidence (by longitudinal studies – over time) and prevalence (by thorough cross-sectional studies). Such studies, if conducted on wild populations, generally require radio-telemetry (hence are labour-intensive) and experienced veterinary support (for health examinations and mortality investigation), and are therefore expensive. Needless to say, it is high time that both State and Federal governments got serious about funding such research on koalas, given our embarrassing lack of knowledge on the topic.

Implications of disease for population survival

Infectious disease may result in a range of impacts on both the individuals affected and the population as a whole. Impacts on individuals may be insignificant, minor, or serious and lifethreatening. Similarly, impacts on populations may be insignificant, minor, or may lead ultimately to extinction. Those impacts are dependent upon factors such as prevalence of infection, incidence of new infection, pathogenicity of the organism, modes of transmission, population dynamics, and genetic diversity, to name a few.

To put it simply:

Impacts on the individual:

- Insignificant
- Minor debility
- Infertility
- Major debility
- Death

Impacts on the population

- Insignificant
- Reduced fecundity
- Population decline
- Increased vulnerability to extinction
- Inevitable extinction

It is our view that both KoRV and *Chlamydia* are highly significant in both their potential impacts on individuals, and on populations. We believe that, in respect of Qld and NSW koala populations, both should be considered critical threats to long-term viability. It is likely that it is only a matter of time before the same can be said of the Victorian and South Australian koala populations.

Our data, as well as that published by other researchers, suggest that prevalence of disease has little to do with habitat quality. It is common dogma in koala conservation circles that "habitat stress" leads to disease; that high levels of disease are largely due to loss of habitat, urbanisation and consequent stress and "crowding" of surviving koalas. We suggest that this is at best an oversimplification, and is certainly not substantiated by hard data. Although some believe that this paradigm promotes the imperative for habitat conservation (which it may well do), it nevertheless implies that if we conserve habitat, the impact of disease will be abated or abolished. Furthermore, it naturally leads to the assumption that disease will not be a threat to

population viability in large habitat fragments. It is our view that this is a dangerous assumption, and probably not true.

Implications of disease for conservation planning and management

Factoring Disease into PVAs

Given the high level of disease in koalas and the prevalence of infertility in female koalas it is crucial that these factors are included in population viability analyses (PVAs). The variability in prevalence of infection and disease between koala populations means that accurate factoring of these into PVA equations requires more thorough assessment of prevalence across metapopulations. As we have mentioned before, many published infection and disease surveys have probably underestimated disease prevalence due to limited veterinary investigation. It is important to note, that, even with ultrasound imaging of the female reproductive tract, early or subtle lesions may not be detected. In other words, a proportion of koalas are probably infertile even though lesions are not apparent using advanced techniques. Disease models used for PVAs must therefore account for this.

Modeling for the impacts of KoRV are somewhat more difficult: the organism is at 100% prevalence (for argument's sake); we have no definitive predictors of disease; arguably, the most important condition (AIDS) is often a presumptive and sometimes tenuous diagnosis; the molecular epidemiology, transmission and pathogenesis of disease are poorly understood; and the impacts, at a population level, have not, to date, been measured. Needless to say, the whole KoRV situation is very worrying from a species conservation point of view, not least because it is difficult to model.

Controlling KoRV and Chlamydia impacts

At the individual level, clinically controlling the impacts of KoRV, in terms of disease production or progression has not been attempted to our knowledge. Feline Leukaemia Virus (FeLV) infection in cats, which causes a similar constellation of clinical consequences in cats as KoRV infection of koalas, is a good example for comparison. Control of FeLV infection in cats relies on prevention by vaccination, and removal of persistently infected cats from situations which allow exposure of uninfected cats. Treatment of persistently viraemic cats with the anti-retroviral drug AZT/zidovudine (Retrovir®) or interferon-α has been shown to have some clinical benefit experimentally. Otherwise, treatment of such cats relies upon treatment of opportunistic infections and other measures to prevent these infections from occurring. Whether these benefits can be translated to koalas that are viraemic with, or affected by KoRV remains to be tested, and does not realistically provide solutions to conservation issues at this stage. Whether vaccination of koalas can reduce the incidence of disease (in infected animals) or

prevent infection (of uninfected animals) are certainly topics worthy of research prioritisation, because they may have some benefits in terms of conservation at the population level.

At present, one interesting hypothesis that remains to be tested is: that female koalas with high KoRV titres tend to give rise to offspring that also have high KoRV titres; and that the converse is also true. If this is proven to be true, and the assumption that high KoRV titres are associated with increased risk of disease is also true, then reduced incidence of KoRV-associated disease could be achieved by selectively breeding female koalas with low KoRV titres. The option of breeding KoRV-free koalas (at least in Qld and NSW) is an opportunity that appears to have passed us by some time ago.

Control of chlamydial infection in individual koalas is somewhat more effective: the infection can be essentially eliminated by appropriate antibiotic therapy (as it can in other species), but the actual pathology itself is more difficult, and sometimes impossible to treat. Consequently, many koalas affected by chlamydiosis are euthanased (or should be), due to the chronic and permanent nature of their pathology. An important confounding issue in the treatment of chlamydiosis in koalas is our inability to meaningfully assess their KoRV disease status (particularly with regard to immune function), which almost certainly has effects on their chlamydial disease status.

Treatment of closed populations of koalas with antibiotics, with the intent of eliminating infection from the population, is certainly hypothetically possible. The time when such management interventions are required for conservation (and not just considered frivolous suggestions) may soon be upon us. Development of a vaccine to prevent chlamydiosis in koalas is the subject of a current research project. If successful it may provide an additional tool for control of chlamydial disease at the individual and population level.

Although some of the island populations of koalas may be free of infection with KoRV or *Chlamydia* at present, the reliance on these populations alone, for preservation of the species, comes with the hazard of substantial lack of genetic diversity. This has already manifested in the high prevalence of congenital abnormalities, and could certainly result in a reduced ability to respond to an incursion of either pathogen in the future. For comparison, low genetic diversity in Tasmanian devils is suggested to be important in their inability to defend against the facial tumour cells, which in many respects is an "infection" (Siddle *et al.*, 2007).

Priorities for research and funding

In summary, some of the tools that we may have available to control infection and/or disease in both individuals and populations include:

1. Vaccination

- 2. Treatment/elimination of infection
- 3. Selective breeding for disease resistance

All require a significant investment in research time and funding, and a substantially improved understanding of the epidemiology of, and interactions between, the infections which they seek to control. To date, the level of funding, and therefore our state of knowledge, has been quite poor, given the iconic status of the animal and the magnitude of the threat facing its continued existence in the wild.

In comparison to the response to the epizootic of Tasmanian devil facial tumour disease (DFTD) (\$22 million of government funding committed to date) (Lunney *et al.* 2008), our response to the threat of koala disease (particularly KoRV-associated disease) has been minimal. Some of the reasons probably include:

- 1. That disease in koalas has been recognised for years (over a century, in fact), and it is almost accepted as "part of being a koala".
- 2. That DFTD causes dramatic and overt pathology, and has spread rapidly; in contrast, KoRV-associated disease is insidious and often overlooked even with veterinary assessment.
- 3. Severe habitat impacts on remnant koala populations are masking the impacts of KoRV and Chlamydia.
- 4. Regulatory authorities in Queensland and New South Wales are largely ignorant of the threat, mainly due to the poor level of veterinary support and disease surveillance provided to key koala rehabilitation centres.
- 5. The koala is still geographically widespread, and in some areas "over-abundant".

Whatever the reasons, our current poor understanding of the real impacts of disease means that we cannot assume that the koala's hold on existence is any less tenuous than that of the Tasmanian devil, whose extinction from the wild is considered likely (Lunney *et al.*, 2008).

It is important to reiterate that the urgent need for further disease research (and the funding to support it) does not in any way lessen the imperative to apply effort and funding to habitat protection and restoration.

In Summary

The key messages regarding infectious disease in koalas are:

- 1. That it must be considered a key threatening process (that may ultimately contribute to extinction), until proven otherwise;
- 2. That the impact of KoRV on koala population health and survival is unknown, but potentially catastrophic;
- 3. That this potential *drives* the imperative for effective habitat conservation and restoration, rather than detracting from it;
- 4. That State and Federal government understanding and acknowledgement of the potential impacts of disease on the conservation of koalas is poor.
- 5. That there is an urgent need for the application of appropriate effort and government funding to better understand and (hopefully) mitigate the impacts of epizootic infectious disease in koalas.

Finally, we encourage all koala conservationists and carers to use and share your knowledge, statistics and experience to lobby government, unrelentingly, for change.

Epilogue

At the time of writing, the Australian Koala Foundation has an application for federal listing of the Koala Coast koala population as vulnerable under the Federal *Environment Protection and Biodiversity Conservation Act*. Letters in support of this application, expressing concern also for your local koala populations, can only help the cause, so we urge you, respectfully, to put pen to paper and let them know what is happening at the coalface.

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APPENDIX 1

	Ko	Australian Wildli Pala Examina	-	at a	
		ala Lammi	ation she		
Animal Details			See Accession	n Form (a	or complete details below)
Animal's Name		Ac	ccession No		
Gender	Male Fer	male 🗌 Intersex 🛮 QI	PWS Form No		
Re-Admission	No	Yes (previous Acces	sion No)
Rescuer Details					
Rescuer Name		Af	filiation/Group		
Rescuer Address			elephone (home)		
(optional)		Te	elephone (mobile)		
Email Address					
Caller Details					
Caller Name		Te	elephone		
Reason for calling					
Rescue Details		<u>, </u>			
Date of Rescue			Time of Rescue		AM/PM
Exact location of rescue					
Grid Reference			LGA	Ą	
Reason for Rescue					
Position of koala	In tree		In captivity		
Identifying Feature					
	□ No	□ Vas. Tag Na			Left Right
Ear Tag		Tes - rag No.			Left Right
Microchip	☐ No	Yes – No			
Other identifying feature	s:				
Summary of Diagno	oses:				
1.					
2.					
3.					
4.					
	RV Suspicion	n	Chlamyd	lia	Other
? Aids		astic anaemia	Eyes	IU	Trauma
Lymphoma		ner Neoplasm	Urinary / Rena	al	Orphan
Plantar hyperkeratosi			Respiratory		Healthy
Leukaemia	□		Reproductive		
Myelodysplasia					
Initial Outcome:					
Dead on arrival		Euthanased		Admi	t to Hospital
Died during examinat	ion	Immediate release		Sent 1	to Carer on / / 20
Final Outcome:					
Euthanased on /	/ 20	Sent to carer on	/ / 20		sed on / / 20
Died on /	/ 20	Permanent care @		Trans on	ferred to/ / 20
D-1 D-421-					
Release Details:			Released by:		
Release Details: Release Date:			leleased by.		
	»:		Release authorised	by:	

Demeanour	☐ B.A.R. ☐ Depressed ☐ Excited ☐ Moribund
3ehaviour	Normal Abnormal
Posture	Normal Abnormal
Gait	□ Normal □ Abnormal
Symmetry	Normal Abnormal
Breathing	☐ Normal ☐ Shallow ☐ Rapid ☐ Laboured
Coat	Normal Abnormal
Discharges	□ Nil □ Present
Wounds/Bleedin	g Nil Present
Other lesions:	Nil Present
Abdomen:	☐ Normal ☐ Bloated ☐ Sunken
	Route: i.m. i.v. facemask Tube
Anaesthesia	Maintenance Anaesthetic Agent
Anaesthesia Vital Signs	Maintenance Anaesthetic Agent
	Maintenance Anaesthetic Agent
	Maintenance Anaesthetic Agent
Vital Signs	Maintenance Anaesthetic Agent
Vital Signs Hydration Weight / Age /	Maintenance Anaesthetic Agent Dose Route: i.m. i.v. facemask Tube Mucous Membrane: Pink Pale Cyanotic Red HR Rectal Temp Rectal Temp CRT SpO2 Pulse: Rate SpO2 Rhythm: Regular Occasional arrhythmia Frequent/constant arrhythmia Amplitude: Normal Increased Decreased Tone: Normal Increased Decreased Normal Some Some Some Some Some Some Some Some
Vital Signs Hydration Weight / Age / Body Score	Maintenance Anaesthetic Agent

Musculoskeletal	Normal			
	Head Symmetry Ears Lips Nares Tongue	Normal Abnorm Normal Abnorm Normal Abnorm Normal Abnorm	mal mal mal mal	
Head / Mouth	Teeth Gingiva Fauces Palate/tonsils Pharynx Larynx Cheek Pouches	Normal Abnorm Normal Abnorm Normal Abnorm Normal Abnorm Normal Abnorm Abnorm	mal mal mal mal mal	
Eyes:		LEFT		RIGHT
Periorbital skin: Eyelids: Palpebral fissure Conjunctiva:	Normal Normal Normal Other	Alopecic ☐ Pigmented Other ↓ Scarred ☐ ↑ Proliferated 1 2	3	Normal Alopecic Pigmented Normal Other The control of the
Nictitating: Sclera: Cornea: Opacity: Iris & Pupil: Discharge: Description:	Normal Normal Normal Normal Normal Normal Serces	Prolapsed 1 2 Other Abnormal Mild		Normal Prolapsed 1 2 Normal Other Normal Abnormal Clear Mild Marked Normal Abnormal Nil Serous Purulent Amt: 1 2 3
Schirmer Tear Test (60 secs): Fluorescein Test:	Length	mm		Lengthmm
Coat:	Colour: Structure: Texture: Other:	Light Grey Normal Normal	Brow Spars Grea	se Clumped/irregular
Skin:	Normal	Abnormal		
Lymph Nodes	Rostral mandibul Facial Mandibular Superficial cervic Axillary Inguinal Notes	Normal L R	Enlar Enlar Enlar Enlar	rged L R

General Physical Examination (continued): Abdominal Fill Normal Abnormal Stomach Fill ☐ 1 – (empty) ☐ 2 (½ full) 3 (full) Stomach Consistency Normal (firm) ☐ Soft ☐ Bloated ☐ Abdominal Distal colon Pellets Empty/ pellets not palpated Palpation Proximal colon/caecum Normal Abnormal Notes /Other abdominal lesions: Clavicle: Normal Abnormal Chest Palpation: Ribs: Normal Abnormal Normal Left Forearm Abnormal Left Paw/digits: Normal Abnormal Left Hindleg Normal Abnormal Left Foot/digits: Normal Abnormal Limbs and Joints Normal Right Forearm Abnormal Right Paw/Digits Normal Abnormal Right Hindleg Normal Abnormal Right Foot/digits: Normal Abnormal Normal Abnormal Scrotum / Pouch Active Inactive Abnormal Scent Gland/ Mammary Glands Normal Abnormal Cloaca/Clitoris/ Penis (circle score) Rump stained: 0 (no staining) 1 (mild) 2 (marked) Rump wetness: 0 (dry) 1 (damp) 2 (dripping wet) Cloaca: 0 (normal) 1 (red/inflamed – slightly protruding) 2 (marked protrusion – no ulceration) 3 (2 + ulceration/pseudomembrane) Dirty Tail Score Rump: 0 (normal) 1 (inflamed skin – no decubital ulcers) 2 (decubital ulcers or erosions from urine scalding) Dysuria: 0 (nil/not observed) 1 (apparent discomfort when urinating and/or vocalization) Total Score: Other Koala Details, History or Previous Treatments:

Procedures Perfori	med:
CLINICAL PATHOL	OGY
Blood Stain:	☐ In-House ☐ External (Idexx/other) PCV% TSg/litre ☐ Giemsa ☐ Diff-Quick ☐ Other Slide Kept? ☐ Y ☐ N
Bone Marrow Smear:	Stain: Giemsa Diff-Quick Other Slide Kept? Y N Collection Site: Iliac Crest L R Other
Abdominal Aspirat	e Stain: Giemsa Diff-Quick Other Slide Kept? Y N
Faecal Analysis Gross Examination:	Shape Normal Abnormal
Tests:	☐ Float ☐ Wet Prep ☐ Stain
Urinalysis Smear:	Collection Method: Cysto Catheter Free catch Urinalysis Urine Sediment Smear USG
Urinalysis: pH: Pr: Chlamydia (Cleary Rate positives on a scale of 1- the following chart: 4: very strong, 2- vec control 3: strong - vec but < - ve control 2: weak - ve_, easily seen 1: very weak, barely perceptib Radiographs	Test site
Other Diagnostic A	Aids

	Urine/Lume Wall Thickr	en:			١			• • • • • •			
	Wall Thickr		Cle	ar		Flocculent ed	cho		Lumena	l c	ast/other
		Wall Thickness at point of widest lumen (transverse section)									
						LEFT		LU	MEN		RIGHT
	Horizonta	I									
Bladder Vertical											
	Left Diago	nal									
	Right Diag	gonal									
 											
remaie Reproductive											
DICUT	Abnorm	ıal									
							,				
luctive						_		•			
LEET	Abnorm	1al			•••••						
LLII	D Evansi-					ovaminad			or image	••••	
									Ü		
	Frontal plane diameter mm (at widest diameter)										
	1 Torreal plan	ic al	annetel a				(at	VV10			aht
Overall S	tructure:		Normal		Lej		1	$\overline{}$		M	yııı
0 7 6 7 4 11 5	ci docai oi	ı =			al		[Abnormal			
Measure	0							Lengthmm			
								Vert. Diametermr Horiz. Diametermm			
Parenchy					Ti	Normal					
			Hyperec	hoic		Hypoechoic	j				Hypoechoic
	lvis:		Normal		=]	=		L	Dilated
		ዙ				Dilated]	=		L	Dilated
Lesions:		H					[]	Present			
	RIGHT luctive LEFT Overall S Measure Parenchy	luctive Normal Abnorm RIGHT Normal Abnorm LEFT Examing Normal Normal Overall Structure: Measurements: Parenchyma echo: Renal pelvis: Ureter:	RIGHT Abnormal Normal Abnormal Examined Normal Normal Frontal plane di Abnormal Examined Normal Normal Frontal plane di County of the plane o	Normal	Normal	Normal	Normal	Normal	Normal	Normal	Normal