ANSC20003 Topics in Animal Health

THE UNIVERSITY OF MELBOURNE · UNDERGRADUATE LEVEL 2 · SEMESTER 2, 2026

Full guide + ask Sia about ANSC20003

SIDE 1/2 EXAM MAP · MECHANISMS · PATHWAYS · CLASSIFICATIONS · KEY VALUES — ONE A4 NOTE SHEET — Mechanism → evidence → limit

01 · Exam map

COURSE.JSON · CANVAS 2026

35% reports | 15% mid | 50% final

ASSESSMENT	FORMAT	WEIGHT
Two practical reports	750 words each	35%
Mid semester exam	Week 7	15%
End of semester examination	2 hours	50%

Final: 120 marks in 120 minutes. Thirty 1-mark MCQs (A–D) + 90 marks of medium/long answers worth 3–10 marks; 15 minutes reading time first.

PERMITTED MATERIALS *"You can bring one double-sided A4 sheet of printed or hand-written notes to the exam, a non-programmable calculator and up to 2 sheets of blank paper." ANSC20003 End of Semester Exam Information page, Canvas 2026.*

02 · Disease answer frame

CH 1

SICK ANIMAL

- **CAUSE** cause/event
- **SIGNS** animal + record
- **DIAGNOSIS** sample + test
- **TREATMENT** this animal now
- **PREVENTION** stop next case

Five fixed headings are both recall scaffold and completeness check.

Before naming disease: Is this disease rather than environment, stress or anxiety? Is it serious for present/future quality of life? Can it wait hours or days? Build a ***differential diagnosis***; identify systems before diseases; common problems occur commonly.

GROUP PROBLEM · WRITE IN ORDER

```
Establish issue → prepare → differential list → describe outbreak → evaluate diagnosis → control + prevent → communicate.
```

03 · Inflammation clock

CH 1

IMMEDIATE seconds–days · fluid · leak · wall off → **DELAYED** days–weeks · cells · vessels · collagen → **OUTCOME** resolution · fibrosis or granuloma

Phase first, then mechanism: fluid → cells → resolution or persistence.

Five signs: redness, swelling, heat, pain, loss of function. Disease damage may be physical, physiological, chemical or immunological; observed signs combine that damage with inflammation, immune stimulation, systemic change and healing.

WOUND PHASE	TIMING	DOMINANT WORK
Fluid	<2 d	Scab; wall off; epithelium moves
Granulation	>10 d	Vessels, fibroblasts, rising collagen

04 · Bacterial wall → result

CH 2 · PRAC 1–2

GRAM + thick peptidoglycan — purple

GRAM − outer membrane — pink

Thick wall retains crystal violet; thin wall loses it and takes counterstain.

GRAM SEQUENCE · WHERE IT BITES

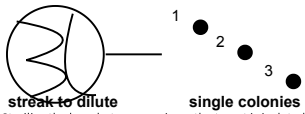
```
Dry → heat-fix → crystal violet → iodine → decolourise → safranin. Over-decolourising can report Gram-positive cells as Gram-negative.
```

STRUCTURE	JOB / CONSEQUENCE
Fimbriae	Adhesion, colonisation
Pilus	DNA transfer by conjugation
Flagellum	Motility
Capsule	Shielding; resists drying
Endospore	Survives heat, chemicals, drying, radiation

Commensal means normal flora in a healthy animal. Changed health or entry through a wound may turn the same organism opportunistic; do not assume it acquired a new trait.

05 · Culture decides the claim

CH 2 · CH 12



Sterilise the loop between regions; the target is isolated colonies.

METHOD / MEDIUM	WHAT IT ANSWERS
Streak plate	Isolation
Spread plate	Counting
Pour plate	Aerobic + anaerobic growth
General purpose	Broad growth
Enriched	Fastidious growth
Selective	Who grows
Differential	What growth looks like

Identification needs a pure culture. Describe colony diameter, pigmentation, form, elevation, margin, surface, opacity and texture; then interpret the medium. A phenotypic key gives a *presumptive* identity; further biochemical testing or sequencing confirms.

NAMED PLATE	READ IT AS...
Mannitol salt	Salt tolerance; mannitol fermentation
MacConkey	Enteric selection by bile salts; lactose fermentation; pink = lactose fermenter
Aesculin sheep blood	Enriched growth; haemolysis + aesculin hydrolysis

06 · Virus structure predicts control

CH 4

	ENVELOPED	NON-ENVELOPED
Outside	Host-derived lipid + glycoproteins	Protein capsid
Stability	Labile	Stable
Sensitive to	Detergent, acid, drying, heat	Resistant to all four
Spread	Moist conditions; close contact	Survives on surfaces
Exit	Budding or lysis	Lysis or export

Viruses are ***obligate intracellular parasites***: natural host, laboratory animal, embryonated egg, tissue/organ culture or cell culture—not broth. An unchanged monolayer is not proof of absence: infection may be non-cytopathic, persistent or latent.

1 ATTACH	receptor → range/tropism
2 ENTRY	endocytosis or fusion
3 UNCOAT	genome released
4 COPY	nucleus or cytoplasm
5 EXIT	assembly → budding or lysis

The replication stage names the biological decision and likely intervention point.

07 · Infection ≠ disease

CH 4–5

PRIMARY acute signs — **REACTIVATE** stress/illness

LATENCY · no signs

days → months or years

Latent infection persists without continuous shedding or clinical signs.

- **Direct test:** antigen/protein, nucleic acid or particle; answers whether target is present in that sample now.
- **Biological:** growth in living cells; visible change is cytopathic effect.
- **Indirect:** host antibody/cell-mediated response; exposure, not necessarily current infection.

Sampling follows pathogenesis: entry → spread → target damage → shedding occur in different places and times. The right test on the wrong sample or at the wrong time can be negative in an infected animal.

Herpesvirus: primary infection → latency → reactivation. Spillover severity may reflect a novel host lacking co-evolved defences, not a universally more virulent virus. Recombination requires **two live, replicating genomes in the same cell at the same moment**; an inactivated preparation cannot participate.

WILDLIFE CONSTRAINT	EFFECT ON EVIDENCE
Sick animals are predated	Clinical disease is under-observed

08 · Parasite map

CH 6

CLASS	RECOGNITION / CONSEQUENCE
Protozoa	Unicellular, 2–100 µm; stained smear
Helminths	Multicellular worms: cestodes, trematodes, nematodes
Arthropods	Ectoparasites: ticks, mites, fleas, lice, flies

Harm mechanisms: mechanical tissue damage; nutrient loss; damaging host inflammation; reduced appetite. Only the first is direct parasite action.

DIRECT

definitive adult + sex → next definitive

INDIRECT

definitive → intermediate develops → definitive

Remove a required intermediate host to break an indirect cycle.

HOST	MEANING	CONTROL LOGIC
Definitive	Adult + sexual reproduction	Source of reproductive stages
Intermediate	Immature/developing stage; required	Removal breaks cycle
Paratenic	Carries; no development	Removal lowers transfer, does not break cycle

Control means finding the cycle's vulnerable step, not merely killing adults: transmissible larvae or cysts left in the environment can reinfect treated animals.

09 · Three clocks

CH 6–7

infection — eggs found — signs

PRE-PATENT

INCUBATION

PATENT · test works

Illness can precede egg detection; a negative early faecal test may fit infection.

WHITLOCK UNIVERSAL COUNT

```
EPG = (chamber 1 + chamber 2) × factor
Sheep factor 30 · cattle factor 15.
Two chambers = 1 mL of a 60 mL suspension; divide by 2 g sheep or 4 g cattle faeces.
```

Mix side-to-side; load immediately from the middle. Count strongyle eggs separately. FEC cannot name genus (use faecal culture) or show drench efficacy (use paired pre/post counts).

INTERPRET ONLY AFTER ASKING...	WHY
Age + physiological state	Susceptibility and burden tolerance differ
Host + worm species	Egg output and harm differ
Nutrition + clinical signs	Same count can carry different consequence

10 · ELISA: target decides design

CH 8

INDIRECT	SANDWICH
coat: antigen	coat: capture Ab
sample: antibody	sample: antigen
labelled anti-Ig	detection Ab
+ substrate	+ substrate

Indirect asks "has the host responded?"; sandwich asks "is antigen here?"

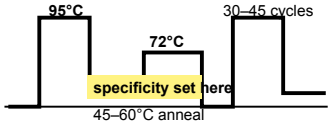
Positive serology establishes exposure to the pathogen or something antigenically similar—not current infection, shedding or exposure date. Primary exposure: IgM first, then some IgG. Secondary exposure: faster, higher, longer IgG. The first ~1–2 weeks may be a ***blind window***.

ANTIBODY	STRENGTH	TRADE-OFF
Monoclonal	One epitope; consistent	May miss changed/hidden epitope
Polyclonal	Many epitopes; robust binding	Batch variation; more cross-reaction

Sandwich capture and detection antibodies must bind different epitopes; monoclonals make that separation controllable. Choose assay from infection dynamics, target, sample, detection window and field/laboratory setting.

11 · Amplification + controls

CH 9



Denature 95°C → anneal 45–60°C → extend ~72°C; repeat 30–45×.

PART	JOB / INTERPRETATION
Template	Target from chosen sample
Primer pair	Defines ends; sets specificity
Thermostable polymerase	Builds new strand
Nucleotides	Building blocks
Buffer + Mg^{2+}	Reaction conditions
Positive control	Chemistry + primers worked
No-template control	Must be blank; reveals contamination

Controls first, samples second. A band of expected size supports amplification of something compatible with the primers; it does not alone identify the organism. Confirm by sequencing or, for a short product, melt analysis. More cycles increase signal *and* non-specific amplification risk.

OBSERVATION	VALID CONCLUSION / RESPONSE
Positive control blank	Run failed; sample negatives uninterpretable

12 · Defence layers

CH 10

BARRIERS · continuous
skin/mucosa · mucus/cilia · secretions · flora

INNATE · minutes–hours
PRRs · inflammation · phagocytes · C′

ADAPTIVE · several days first exposure
specific B/T · clonal expansion · memory

Innate holds the line while specific adaptive immunity develops.

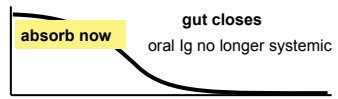
READER	WHAT IT SEES	EFFECTOR LOGIC
B-cell receptor	Accessible shape on intact antigen	Antibody acts outside cells
T-cell receptor	Peptide displayed in MHC	Requires processing/presentation
CD8 T cell	Infected-cell peptide	Kills infected cell
CD4 T cell	Presented peptide	Cytokines; activates macrophages

Pattern-recognition receptors detect conserved microbial structures or host damage and trigger inflammation. Dendritic cells capture antigen in tissue and activate naïve T cells, linking innate and adaptive arms. Adaptive properties: specificity, diversity, memory and tolerance.

IMMUNITY	SOURCE	MEMORY / DURATION
Active	Recipient response after infection/vaccination	Memory; delayed onset
Passive	Ready-made maternal or administered antibody	No recipient memory; temporary

13 · Newborn timing

CH 11



Absorption falls sharply from ~6–24 h; gut closure completes in the first day.

FAILURE	HISTORY CLUE
Production	Dam produced poor/insufficient colostrum
Ingestion	Newborn did not drink enough
Absorption	Adequate intake, low serum Ig

Cattle transfer no antibody prenatally; calf systemic immunoglobulin therefore depends on colostrum. Colostrum Ig is absorbed systemically; milk antibody stays in the intestine as local ***lactogenic immunity***. Maternal antibody protects but suppresses the neonate's own antibody synthesis, so vaccine schedules use a series across ages rather than one predicted date.

EXAM HINGE *Route follows age: oral supplementation only while absorption remains possible; after closure, deliver immunoglobulin directly to circulation and re-measure to confirm transfer.*

ANSC20003 Topics in Animal Health · Drills & Traps

THE UNIVERSITY OF MELBOURNE · WORKED APPLICATIONS · FAST-RECALL CONTRASTS

Full guide + ask Sia about ANSC20003

SIDE 2/2 · WHICH-IS-TRUE · WORKED APPLICATIONS · PRACTICAL CONTROLS · SHORT-ANSWER FRAMES

ONE MARK ≈ ONE MINUTE

Name → apply → limit

14 · Which statement is true?

FAQ · ALL CH

TEMPTING FALSE CLAIM	TRUE CONTRAST
Fever proves infection.	Heat, transport, anxiety and pain can also raise temperature.
All normal flora are harmless.	A commensal can become opportunistic when health/site changes.
Gram colour is cosmetic.	It reports wall structure and narrows identification.
Selective = differential.	Selective controls who grows; differential reveals what growth does.
No cell-culture change means no virus.	Some infections are non-cytopathic, persistent or latent.
Species-specific means no spillover.	Captive proximity can expose a novel host.
Paratenic = intermediate.	Only the intermediate host is required and supports development.
Negative faeces excludes parasitism.	Signs may begin before the patent period.
Low FEC means no treatment.	Interpret age, physiology, host, nutrition, worm species and signs.
Positive serology means current shedding.	It establishes exposure, not timing or current infection.
Expected-size band proves identity.	Confirm; controls and primer scope limit the claim.
High antibody clears any infection.	Antibody cannot reach an intracellular pathogen.
Milk gives systemic immunity.	After closure, milk antibody protects locally in the intestine.
Final weight = feedlot performance.	Use gain relative to induction weight and days fed.

WHAT MUST BE SHOWN?

CURRENT?	EXPOSURE?	RELATED?
direct target	antibody	isolate/strain

check sample · time · controls

Start with the claim, then choose target, sample, time and controls.

FAST ELIMINATION RULE

```
If an option jumps from an observation to a stronger claim, ask which missing link would be required: viable isolate, confirmatory sequence, paired titre/count, appropriate timepoint, or valid control.
```

Absolute words are warning signs. “Always,” “never,” “proves,” “all animals” and “rules out” are usually wrong where sampling, timing, host state or a population decision intervenes. Match the option to the exact biological level: molecule, organism, animal, herd/flock or production system.

When two options are true in isolation, prefer the one that answers the stated animal, sample and timepoint.

15 · Worked: faecal egg count

CH 7 · PRAC 3

Given: sheep; two Whitlock chambers contain 14 and 18 strongyle eggs.

CARRY THE ARITHMETIC TO THE END

```
Count = 14 + 18 = 32 eggs
Sheep factor = 30
EPG = 32 × 30 = 960 eggs/g
```

2 chambers = 1 mL	suspension = 60 mL	sample 2 g sheep

×60 ÷2 g = ×30 per counted egg

cattle uses 4 g → factor 15

The factor is chamber volume and sample mass arithmetic, not a species correction.

Supports: a high individual count under the published bands. **Does not establish:** genus/species—strongyle eggs look alike; culture larvae. Nor does one animal settle the flock decision; sample more widely if others are lower.

EPG	PUBLISHED CONSEQUENCE BAND
0–100	Few situations warrant drench
101–200	Still low; trigger only in some programmes
201–500	Production loss/scouring may occur
501–1000	Entering high range; significant loss
1001–2000	High; clinical signs may be obvious
>2000	Severe loss; deaths possible

For drench efficacy, pair pre-treatment with 10–14-day post-treatment counts. Effective means at least about 95% reduction, ideally ≥98%; below this indicates resistant worms.

REPORT IN FOUR CLAUSES

```
Result + unit → place against consequence band → name what FEC cannot establish → state flock/sample/context qualifier before action.
```

Do not turn a risk band into a blanket drench instruction. A flock can acquire serious burden faster than its count rises to meet it. Conversely, one high individual may not represent the flock. The correct action can be repeat sampling, broader sampling, culture for genus or paired reduction testing—each answers a different uncertainty.

UNKNOWN	NEXT METHOD
Is the count repeatable?	Repeat well-mixed chamber/sample
Which strongyle genus?	Faecal culture → identifiable larvae
Does this represent the flock?	Broader individual sampling
Did the drench work?	Paired count after 10–14 d

ARITHMETIC CHECK *Write the chamber-specific factor before multiplying, then finish with eggs/g.*

16 · FEC method traps

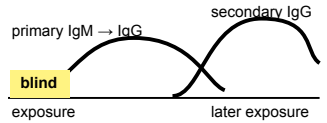
CH 7

17 · Worked: test the timepoint

CH 8–9

INDIRECT ASSAY · DAY 4 OF ILLNESS

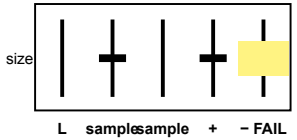
The animal is inside the roughly 1–2 week antibody blind window. A negative is expected with or without infection, so it is not evidence against disease. It does support no earlier exposure that left detectable antibody. Use a direct antigen or molecular method on the sample where the pathogen should currently be found.



Negative serology inside the response window cannot exclude current infection.

AMPLIFICATION · BAND IN WATER CONTROL

The whole run is invalid. Template entered the no-template reaction; the sample band cannot be separated from carry-over. The positive control only shows chemistry and primers worked. Discard results, find the source, use fresh materials and repeat—do not salvage lanes.



Read + and − controls before any sample; a band in − invalidates all lanes.

18 · Test answer scaffold

CH 8–9 · CH 13

FOR ANY DIAGNOSTIC PROCEDURE

```
1 What target/information?
2 Why this sample + time?
3 What do controls establish?
4 Advantage?
5 Limitation?
6 Confidence and confirmatory step?
```

Agglutination: read saline first; clumping in saline is autoagglutination, so the isolate is uninterpretable. **Gel:** higher voltage is faster but resolves worse; size by comparison with a known ladder, not by visual measurement.

IF THE QUESTION ASKS...	USE...	THEN LIMIT IT
Past exposure	Indirect antibody assay	Not current infection/shedding
Current antigen	Sandwich assay	Needs target in that sample now
Current nucleic acid	Amplification	Not viability; contamination risk
Viable organism	Culture	May be slow; no growth can be uninformative

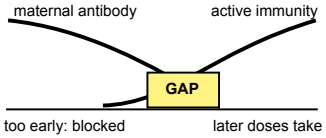
19 · Worked: newborn at 20 h

CH 11

Given: foal serum immunoglobulin below adequate threshold at 20 h. The timing is after absorption is complete, so the value reports transfer outcome. Oral colostrum works only in roughly the first 12–15 h; after gut closure it remains intestinal.

DECISION

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20 h + low serum Ig → intravenous donor/commercial plasma → re-measure Ig. Route bypasses the closed gut; re-test verifies rather than assumes transfer.
```



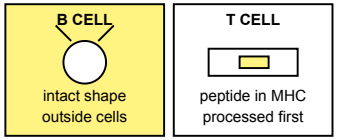
Maternal antibody can be too low to protect yet high enough to block vaccination.

STATEMENT	CORRECTION
Milk replaces colostrum.	Milk Ig acts locally in gut; colostrum can supply systemic Ig before closure.
Low Ig tells the failure type.	History separates production, ingestion and absorption failure.
Vaccinate immediately.	Maternal antibody may block response; use a dose series.

20 · Worked: antibody but no clearance

CH 10

Given: intracellular pathogen + high antibody titre + persistent infection. Antibody is large and hydrophilic and cannot cross cell membranes, so it reaches exposed extracellular epitopes, not the intracellular pathogen.



B cells read intact accessible shapes; T cells read processed peptide in MHC.

Needed: cell-mediated immunity—CD8 cytotoxic T cells kill infected cells; CD4 helper T cells release cytokines and activate macrophages. State location before choosing immune arm.

Extracellular, inside a phagocyte and inside an infected cell are three locations with different immune solutions.

TRAP → FIX *A high antibody titre shows binding, not access. First locate the pathogen: extracellular → antibody can act; intracellular → choose cell-mediated effectors.*

22 · From sample to named pathogen

CH 12–13 · PRAC 1–4

HISTORY	animals · signs · change
SAMPLE	tissue where pathogen should be
STAIN	broad group in minutes
CULTURE	selective → biochemical ID
LINK	serotype / sequence / PCR

Each step narrows the list, costs more, and supports a different claim.

Zoonotic outbreak logic: sheep abortion + human gut illness after contact + dead scavenging bird are constraints; the candidate must explain all three. For abortion, products of conception may contain more organism than a dam that has cleared it.

STAGE	SHOWS	CANNOT ALONE SHOW
Gram stain	Broad wall/morphology group	Species
Selective culture	Viable organism with growth traits	Definitive identity
Biochemical panel	Profile pointing to species	Outbreak relatedness
Serotyping	Surface-antigen relation	Valid result if saline autoagglutinates
Molecular test	Target nucleic acid	Viability; isolate for serotyping

SHORT ANSWER *Separate identity from relatedness. A rapid molecular screen may guide immediate movement restriction while culture incubates, but removing culture removes the isolate needed to link cases.*

MILK-SAMPLE SEQUENCE	CRITICAL DETAIL
Streak three plates	Successive dilution to isolate
Incubate	Aerobically at 37°C overnight
Describe colonies	Observe first; interpret each medium second
Gram smear	One well-isolated colony; oil-immersion image
Catalase + key	Avoid agar; reach presumptive identity

23 · Contradictory practical results

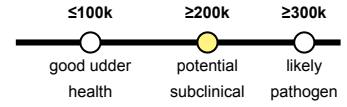
CH 12

Reported Gram-negative rods grew on salt-selective medium but not enteric-selective medium. Both plates point away from the microscopy. Most likely: over-decolourisation stripped crystal violet from Gram-positive cells. Repeat a smear from the **same single colony** with a two-second decolourisation; reconcile before naming.

24 · Worked: rising milk-cell count

CH 3

Given: bulk tank somatic cell count rises from ~150,000 to ~260,000 cells/mL in two months; no clinical cases.



cells per mL · continuous signal, not diagnosis

The count rises before milk looks different; cross 200,000 → investigate.

Interpretation: 260,000 exceeds the 200,000 potential-subclinical threshold but remains below 300,000, associated with likely significant-pathogen infection. No visible illness fits subclinical mastitis.

Next: individual-cow counts identify contributors; isolate/strain work chooses the control pathway.

PATHOGEN GROUP	TRANSMISSION	CONTROL FOCUS
Contagious	Cow-to-cow at milking	Milking order, teat disinfection, hygiene, machine function
Environmental	Bedding, yard, surroundings	Bedding, drainage, teat condition between milkings

25 · Feedlot decision frame

CH 14

TAIL ACROSS REPEATED CONSIGNMENTS

MODIFIABLE	NOT NOW
health · competition bunk · ration · pen	genetics · sex · age real, but fixed

Recurrence across consignments points first to the repeated routine or facility.

Ask: Were lambs drenched, vaccinated and drafted by liveweight? Do the same pens underperform? Competition, ration, acidosis and pen conditions widen spread.

DESCRIBE, THEN COMPUTE

```
Check symmetry → one/multiple peaks → outlier or group → choose summary.
Average daily gain = (final weight - induction weight) ÷ days on feed.
```

Final weight confounds starting size with performance. One extreme is an outlier, not yet a tail; multiple peaks suggest groups. The working rule treats 3 SD from the mean as highly unusual.

26 · Long-answer construction

COURSE STUDY STRATEGY

EVERY MARK-BEARING CLAIM

```
Name mechanism/class → apply to animal, sample, time or number → limit the claim → act with a reason.
```