0 · Exam Blueprint READ FIRST

★ FOOD90023 is **70% written exam**: a 1-hr mid-semester exam (20%, closed book · calculators only, SAQ + MCQ) and a 2-hr final (50%, ~7 short-answer/essay Qs). Plus 6 fortnightly quizzes (10%) + a 1000-word practical report (20%).

SAQs reward **full sentences + a named example + a number**. The MCQ section mirrors the quizzes. The coordinator *recycles* questions across the MTE and final.

Guaranteed-recurring SAQs: bacteriophages (define + lytic/lysogenic + food uses); the swollen-can scenario; infection vs intoxication + exotoxin classes; bacterial vs fungal spores; 3 gene-transfer mechanisms; malolactic fermentation + its inhibitors; the growth-maths calc $N=N_0 \cdot e^{\wedge}(\mu t)$; conventional-ID limitations; validation vs evaluation.

SIA → *Two reflexes that win marks: always name the organism + a number (don't write "bacteria" – write "spore-forming anaerobic Clostridium"), and on calculations show the working with logs – method marks survive a slipped final figure.*

1 · Classification & Taxonomy LEC 2

Taxonomy = classification (grouping by similarity/phylogeny) + nomenclature (naming) + *identification* (placing a new isolate). Hierarchy: Domain > Kingdom > Phylum > Class > Order > Family > Genus > Species > Strain.

Binomial (Linnaeus): *Genus* (cap) + *epithet* (lower), italic; e.g. *Staphylococcus aureus* → *S. aureus*. Strains carry the safety load: *E. coli* O157:H7 (O=cell-wall, H=flagellar antigen) is a pathogen; most *E. coli* are harmless.

Prokaryote (Bacteria, Archaea): no nuclear membrane, single circular chromosome, 70S ribosomes, no organelles. **Eukaryote** (fungi, protozoa): true nucleus, organelles, 80S.

Trap: classification = the scheme; identification = the process. Archaea ARE prokaryotes but **lack peptidoglycan** & their transcription resembles eukaryotes.

1b · Microbial Groups in Food NAME ONE EACH

GROUP	KEY TRAIT · EXAMPLE
Bacteria	peptidoglycan wall, binary fission · most pathogens
Archaea	no peptidoglycan · halophiles (salt foods)
Fungi	chitin wall, eukaryote · *Aspergillus*, yeasts
Protozoa	eukaryote · *Toxoplasma*, *Cryptosporidium*
Viruses	not a cell; contaminate, never multiply · Hep A, norovirus
Prions	infectious protein, no nucleic acid · BSE/CJD
Helminths	parasitic worms · *Taenia*, *Trichinella*

Trap (MTE): "classes of disease-causing microbes" → list across ALL groups, not just bacteria.

1c · Strain & Species WHY IT MATTERS

A **species** is a group of strains sharing many stable properties; a **strain** is a subset differing by minor traits. The strain carries the food-safety load — *E. coli* O157:H7 is pathogenic, most *E. coli* are harmless. Modern classification uses 16S rRNA for phylogenetic relatedness, not morphology alone.

2 · The Bacterial Cell LEC 3 · ANATOMY

Shapes: cocci (spheres), bacilli (rods), spiral (*spirilla*/*vibrio*/spirochaetes); 1–10 µm. **Arrangements:** diplo-, strepto- (chains), staphylo- (clusters), tetrad, sarcina.

STRUCTURE	FUNCTION
Cell wall (peptidoglycan)	rigidity, shape, prevents osmotic lysis
Plasma membrane	permeability barrier; energy (ETC), transport
Capsule / slime	adhesion, antiphagocytic, biofilm, virulence
Flagella	motility (proton motive force)
Fimbriae	short, many → attachment
(sex) pilus	long, few → conjugation/DNA transfer
Ribosomes (70S)	protein synthesis
Nucleoid	single circular chromosome, NO membrane
Plasmid	small circular DNA, independent; resistance/virulence genes

Trap: pili = long & few (conjugation); fimbriae = short & many (attachment). Plasmids are NOT in the nucleoid & replicate independently.

3 · Gram +ve vs –ve Envelope ★ LEC 2/3

The **Gram stain**: crystal violet → iodine → alcohol decolourise → safranin. G+ retain violet (purple); G– stain pink/red.

	GRAM +VE	GRAM –VE
Peptidoglycan	**thick** (multilayer)	thin (periplasm)
Teichoic acid	**yes**	no
Outer membrane	no	**yes + LPS**
Stain	purple	pink
Example	*Staph*, *Bacillus*	*Salmonella*, *E. coli*

Alcohol dehydrates the thick G+ wall (traps dye); it dissolves the G– outer membrane (dye washes out). **LPS (lipid A) = endotoxin** → fever/septic shock on lysis. The G– outer membrane gives bile/detergent tolerance (basis of selective media) + antibiotic resistance.

Trap: teichoic acid = G+ only; LPS/outer membrane = G– only.

3b · Reading the Gram Stain PRAC P4

1. **Crystal violet** — all cells stain purple
2. **Iodine (mordant)** — fixes the CV–iodine complex in the wall
3. **Alcohol/acetone** — the decolouriser; the critical step
4. **Safranin** — counterstain (pink)

G+ thick wall dehydrates & traps the complex → stays purple; G– thin wall + dissolved outer membrane loses it → goes pink. Over-decolourising turns G+ cells falsely G– — the classic prac error. Old/dead G+ cells can also stain unevenly (gram-variable), so always use a fresh log-phase culture.

Endotoxin (LPS, structural, heat-stable, G–) vs exotoxin (secreted protein, often heat-labile, G+ and G–) — see side 2. The G– outer membrane is also why those organisms tolerate bile & detergents, the basis of selective enrichment media for gut pathogens.

4 · Endospores ★ MTE Q6

A dormant, highly resistant **survival** structure formed inside Gram-positive *Bacillus* (aerobic) & *Clostridium* (anaerobic). One cell → one spore (NOT reproduction).

Sporulation (triggered by starvation): DNA replicates → axial filament → septum near a pole → **engulfment** of forespore → cortex + germ wall → accumulate **Ca^{2+} + dipicolinic acid (DPA)** → spore coat → core dehydrates → mother cell lyses, spore freed. **Germination** returns it to a vegetative cell when conditions favour growth.

Resistance basis: (a) dehydrated core, (b) Ca-dipicolinate stabilises macromolecules, (c) thermal adaptation + tough coats/SASPs protecting DNA. Resists heat, irradiation, desiccation, chemicals, time.

Significance: survive cooking & pasteurisation → demand commercial sterility (121 °C botulinum cook). *C. botulinum* in canned/anaerobic foods, *B. cereus* in rice, *C. perfringens* in warm-held meats.

SIA → *Bacterial endospore = survival, 1 per cell; fungal spore = reproductive, many per cell. Compare the resistance mechanisms when asked.*

5 · Yeasts & Moulds LEC 4 · FUNGI

Eukaryotes, chitin wall (not cellulose, not peptidoglycan), absorptive/heterotrophic (no photosynthesis). Modes: saprophytic, parasitic, symbiotic.

Moulds — multicellular; **hyphae** (septate or coenocytic) form a **mycelium**; spores on conidiophores; colonise food *surfaces*. Groups: Zygomycetes (*Rhizopus*), Ascomycetes (*Aspergillus, Penicillium*), Deuteromycetes (asexual only).

Mycotoxins: aflatoxin (*A. flavus*, carcinogen), patulin, ochratoxin.

Yeasts — unicellular ~10–20 µm; reproduce mainly by **budding**; in the *depth* of liquid foods. *S. cerevisiae*: bread/beer/wine; meiosis → 4 ascospores under stress.

Single Cell Protein (SCP): microbial biomass (30–50% protein) from algae/fungi/yeast/bacteria on cheap substrates.

Trap: fungal spores = reproduction (contrast endospore); a fungus reproducing only asexually = Deuteromycetes.

5b · Bacterial vs Fungal Spore ★ THE COMPARISON

	ENDOSPORE	FUNGAL SPORE
Purpose	**survival**	**reproduction**
Number	1 per cell	many per organism
Formed by	*Bacillus*, *Clostridium*	moulds, yeasts
Resistance	extreme (heat 121 °C)	moderate

Yeasts & moulds also de-acidify wine: acid-reducing yeasts (*Schizosaccharomyces pombe*) metabolise malic acid → ethanol + CO_2 (compare MLF on side 2).

5c · Yeast Life Cycle & SCP LEC 4

S. cerevisiae alternates **haploid and diploid** vegetative states; starvation → ascus → meiosis → 4 haploid ascospores; opposite mating types fuse to restore diploidy. Osmotolerant/fermentative yeasts (*Zygosaccharomyces*, *Candida*) spoil high-sugar/brined foods (gas, films).

SCP advantages: short generation time, easy genetic modification, grows on cheap/waste substrates, continuous culture; rich in lysine/threonine + B-vitamins. Sources = algae, fungi, yeasts AND bacteria.

6 · Bacteriophages ★★ MTE+FINAL Q1

A **phage** = a virus that infects bacteria; an obligate intracellular parasite, non-living outside a host. Structure: icosahedral head (genome) + contractile sheath/tail + baseplate + **tail fibres**.

Genome: ssDNA, dsDNA, ssRNA *or* dsRNA — "DNA or RNA, never both." Host-specific via tail fibres recognising receptors (LPS in G–, teichoic acid in G+). A phage infects *to reproduce*, not to sicken.

LYTIC (VIRULENT)

Adsorption → Penetration (inject) → Biosynthesis → Maturation/Assembly → Lysis & Release. Seen as **plaques** on a lawn. Biosynthesis comes BEFORE maturation.

LYSOGENIC (TEMPERATE)

Adsorb → penetrate → **integrate** as a **prophage** in the host chromosome → replicated passively each generation → **induced** later to go lytic. Can carry toxin genes (lysogenic conversion).

Trap: integrated genome = prophage; phages CAN survive pasteurisation/freezing/drying but NOT without a host.

6b · Phages in Food SAQ STRUCTURE

Negative (headline): phages lyse LAB dairy starters (*Lactococcus lactis*, *Strep. thermophilus*, *Leuconostoc*, *Lactobacillus*) → fermentation fails, big financial loss. Sources: raw milk, whey, starter cultures; survive pasteurisation.

Positive uses: biocontrol/biopreservation (anti-*Listeria* phage on RTE meat), **biosanitisation** (clear biofilms), **phage therapy** (AMR alternative).

Advantages: natural, safe to eukaryotes, highly specific (spare commensals), no sensory change.

Disadvantages: contaminate fermentations, narrow host range (need a cocktail), resistance evolves, regulatory hurdles.

6c · Phage Structure · Label It TUT 5

- **Head / capsid** — icosahedral; holds the nucleic acid
- **Collar / fibritins** — between head & tail
- **Contractile sheath** — around the hollow core/tail tube; contracts to inject
- **Baseplate (hub)** — anchors the tail to the host
- **Tail fibres** — recognise host surface receptors (specificity)

Defence: bacteria resist phages via **restriction–modification** systems (cut foreign DNA, methylate self).

6d · Virus vs Prion vs Virion TERMS

Virion = a complete infectious virus particle (coat + genome). **Virus** = the agent/type. **Prion** = an infectious *protein* with no nucleic acid (BSE/CJD). A phage is a virus of bacteria; outside a host it is an inert virion.

6e · Lytic-Cycle Order Trap TUT 5 Q11

The lysing enzyme/protein is expressed **late**, after the genome is replicated. The correct order is adsorption → injection → biosynthesis → maturation → lysis — biosynthesis BEFORE maturation/assembly. A common MCQ swaps these or asks "when is substance X (lysozyme) made?" → answer: late. Temperate phages can also **convert** the host: a prophage may carry a toxin gene the host then expresses (a food-safety link to §13 exotoxins).

7 · Microbial Metabolism LEC 7

Catabolism breaks down → releases energy (→ ATP); **anabolism** builds up → consumes energy. Coupled by ATP & NAD(P)H.

MODE	FINAL E^- ACCEPTOR	ATP/GLUCOSE
Aerobic resp.	O_2	**~38**
Anaerobic resp.	NO_3^-, SO_4^{2-}, CO_2	intermediate
Fermentation	organic (pyruvate)	**~2**

Glycolysis (EMP): glucose → 2 pyruvate, net **2 ATP + 2 NADH**; universal, cytoplasmic, anaerobic first stage. Aerobic resp. then adds the TCA cycle + ETC/oxidative phosphorylation for the big yield.

Trap: fermentation = **organic final acceptor + substrate-level ATP only**, NOT merely "no oxygen." Respiration uses an ETC. Glycolysis alone nets only 2 ATP; the big yield is downstream ETC/oxidative phosphorylation, only when O_2 is present.

7b · Fermentation Products LEC 7 · MLF

- **Lactic** — pyruvate → lactic acid; homo- (mostly lactate) vs hetero- (lactate + ethanol/acetate + CO_2). LAB: *Lactobacillus, Lactococcus, Leuconostoc*. Yoghurt, cheese, sauerkraut.
- **Alcoholic** — pyruvate → acetaldehyde → **ethanol + CO_2** (yeast); beer, wine, bread.
- **Propionic** (Swiss-cheese eyes), **acetic** (vinegar, *Acetobacter*).

Malolactic fermentation (MLF) — Final Q6: LAB (*Oenococcus oeni*) decarboxylate harsh L-malic acid → softer L-lactic acid + CO_2 in wine. De-acidifies, raises pH, softens mouthfeel, stabilises. **Inhibited by:** pH <3.1, high SO_2, low T, high ethanol, nutrient limit, lysozyme, low cell count.

Trap: MLF is decarboxylation (de-acidification), NOT alcoholic fermentation — give the definition AND the inhibitors.

7c · Oxygen / Redox Classes SWOLLEN-CAN CLUE

CLASS	O_2 · EXAMPLE
Obligate aerobe	needs O_2 · *Pseudomonas*
Obligate anaerobe	O_2 toxic · *Clostridium* (cans)
Facultative	either · *E. coli*, yeast
Microaerophile	low O_2 · *Campylobacter*

Vacuum/canned/MAP → low Eh favours anaerobes → gas + spoilage.

7d · Spoilage ↔ Fermentation SAME LOGIC

The same metabolic chemistry drives both **spoilage** (unwanted gas/acid/off-odour) and **desirable fermentation** (controlled, beneficial). Aerobic spoilers (*Pseudomonas*) dominate in air; anaerobes/fermenters dominate in vacuum packs, cans & the gut. Gas (CO_2/H_2) production is what blows a can.

"Fermentative microbes are found wherever electron acceptors run short."

Why anaerobes make fewer ATP: with no O_2 (or other terminal acceptor) feeding an ETC, NADH can only be re-oxidised by dumping electrons onto an organic molecule — so the cell harvests just the 2 substrate-level ATP of glycolysis and discards most of the glucose's energy in the fermentation product — which is exactly the acid/alcohol/gas we exploit in fermented foods.

8 · Gene Transfer ★ FINAL Q5

Three horizontal mechanisms — name each *with its agent*:

- **Transformation** — uptake of naked/free DNA by a competent cell.
- **Transduction** — DNA moved by a bacteriophage (ties to lysogeny).
- **Conjugation** — cell-to-cell transfer (often a **plasmid**) via a sex pilus; donor F⁺/Hfr → recipient.

Significance: spreads **antibiotic-resistance & toxin/virulence genes** through food & gut bacteria (AMR).

9 · Conventional ID LEC 10 · PHENOTYPE

Microscopy/morphology: shape, arrangement, **Gram stain**, spore stain, motility, capsule. **Cultural:** selective/differential media, colony form, haemolysis. **Biochemical:** O_2 need, **catalase**, **oxidase**, glucose fermentation, sugar use (API). **Serological:** O & H antigens.

The ID tree: Enterobacteriaceae = G– oxidase–ve fermenters; *Pseudomonas* = G– oxidase+ve non-fermenter; *Campylobacter* = G– catalase+/oxidase+ curved rod.

Molecular: PCR/qPCR (toxin genes), 16S rRNA sequencing (gold standard ID/phylogeny), PFGE, MALDI-TOF, ELISA. Faster, more specific, detect VBNC/unculturable cells.

9b · Conventional Limits ★ MTE Q5 / FINAL Q4

Why phenotype-based ID fails: **slow** (days); many organisms **unculturable/VBNC**; phenotype **varies with conditions**; subjective; **poor strain-level discrimination**; overlapping biochemical profiles. → Molecular (16S rRNA) gives true phylogenetic relatedness.

9c · Validation vs Evaluation MTE Q2

Validation = does the method measure what it claims, reliably/reproducibly? (sensitivity, specificity, accuracy, detection limit vs a reference). **Evaluation** = is it fit for purpose? (cost, speed, ease, robustness). e.g. a rapid PCR kit validated against standard plate count, then evaluated for routine use.

9d · Microbial Media LEC 6 · PRACS

Defined (synthetic) vs **complex** (undefined). By function:

- **Selective** — suppress unwanted flora (bile salts for G–, MacConkey)
- **Differential** — distinguish by appearance (MacConkey; MRS for LAB; OGYE for yeasts/moulds)
- **Enrichment** — favour a target organism

Plate counts: dilute, plate, count colonies (30–300), CFU/mL = colonies × dilution factor.

Side 1 · Quick Traps MEMORISE

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G+ = thick peptidoglycan + teichoic, no OM
G- = thin PG + outer membrane + LPS
Endospore = survival (1); fungal = repro (many)
Phage genome = DNA OR RNA, never both
Ferment = organic acceptor, ~2 ATP only
Pili long&few · fimbriae short&many
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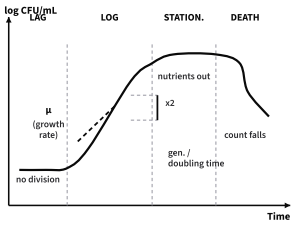
10 · The Growth Curve ★ LEC 8

Bacteria divide by **binary fission** (1 → 2) → exponential growth. Four batch-culture phases:



LAG *(adapt, no division)* → **LOG** *(max rate μ; read generation time here, cells most heat-sensitive)* → **STATIONARY** *(deaths = divisions; spores/secondary metabolites start)* → **DEATH**.

Trap: generation time is defined in the LOG phase; lag length depends on inoculum history, not a fixed value.

10b · Growth Maths ★ FINAL Q7

EXPONENTIAL GROWTH

$N = N_0 \cdot 2^n$ · $N = N_0 \cdot e^{(\mu t)}$
g (doubling) $= t/n \cdot \mu = \ln 2/g = 0.693/g$
$n = (\log N - \log N_0)/\log 2 = 3.3(\log N - \log N_0)$

Worked: $N_0 = 100$, $g = 20$ min, $t = 5$ h ⇒ $n = 300/20 = 15$ generations. $N = 100 \cdot 2^{15} \approx 3.3 \times 10^6$. $\mu = 0.693/0.333$ h $= 2.08$ h^{-1}.

Doubling N_0 only doubles the result; raising μ or t multiplies it by *orders of magnitude* — so **time–temperature control beats lowering initial load**.

SIA → *Use N = Noe^(μt) explicitly, show the logs, and state that the exponent (μ, t) dominates over No – that sentence is the mark.*

10c · Counting Generations LOG METHOD

To get n from counts: **$n = 3.3 \cdot (\log N - \log N_0)$**. e.g. 10^3 → 10^9 CFU/mL is 6 logs ⇒ $n = 3.3 \times 6 \approx$ 20 generations; if that took 4 h, $g = 240/20 =$ **12 min**.

Why preservation targets time & temperature: extending lag (chilling, hurdles) and preventing log-phase growth limits both n and μ — the levers the exponent multiplies.

10d · The Four Phases · Detail WHAT HAPPENS

- **Lag** — adaptation; enzyme/RNA synthesis; cells viable but not dividing
- **Log** — constant max μ, balanced growth; cells most sensitive to heat/antimicrobials
- **Stationary** — growth = death; nutrients depleted, waste & space limit; **sporulation + secondary metabolites** begin
- **Death** — viable count falls exponentially

Predictive models (col 5) fit these phases as λ (lag), μ_max (slope) & N_max (asymptote). The log phase is the danger window: cells divide fastest, so spoilage and pathogen risk climb most steeply here. Growth is "balanced" in log phase, and cells are also most heat- and antimicrobial-sensitive then — the best moment for a kill step.

11 · Factors · Temperature EXTRINSIC · MASTER

Every microbe has min/optimum/max ("cardinal") temps; classed by optimum:

CLASS	RANGE (OPT)	EXAMPLE
Psychrophile	~5 to 20 (~15)	cold spoilers
Psychrotroph	grows at 0–4 °C	*Listeria*, *Pseudomonas*
Mesophile	20–45 (~37)	most pathogens
Thermophile	45–70 (~55–65)	thermoduric

Danger zone ≈ 4–60 °C. Trap: refrigeration only *slows* mesophiles — it does NOT stop psychrotrophs like *Listeria*; refrigeration is not a kill step. **Thermoduric** organisms survive (but don't grow at) pasteurisation temps; **thermophilic** spore-formers cause flat-sour spoilage in canned goods held warm.

11b · Factors · pH & Water Activity INTRINSIC

pH — most bacteria optimum near neutral; pathogens generally need **pH > 4.6** (the basis of "low-acid foods" needing the botulinum cook). Yeasts/moulds tolerate lower pH; pH 4.6 = the *C. botulinum* safety threshold. **Water activity (a_x)** — available water, 0–1; lowered by salt/sugar/drying:

a_x FLOOR	GROUP STILL GROWS
> 0.90–0.91	most bacteria / *C. botulinum*
~0.86	*S. aureus* (salt-tolerant)
~0.80	most moulds
~0.60	xerophiles/osmophiles (floor)

Other intrinsic factors: nutrients, **redox (Eh)/O_2**, natural antimicrobials (lysozyme), physical barriers. Trap: dried foods spoil by mould, not bacteria; *S. aureus* is unusually salt/low-a_x tolerant — so it grows in cured meats & salty foods that exclude most competitors.

11c · Hurdle Concept SYNERGY

Combine several **sub-lethal** factors — mild heat + low pH + low a_x + preservative + low T + MAP — so no single hurdle is harsh but together they prevent growth while preserving quality.
e.g. fermented sausage = a_x↓ + pH↓ + nitrite + smoke.
Trap: hurdle = synergy of mild factors, NOT one strong treatment. Each hurdle stresses the cell's homeostasis; combined, they overwhelm its repair/repair-energy budget before any single one would, so milder individual treatments keep the food fresher.

11d · Intrinsic vs Extrinsic CLASSIFY

Intrinsic = properties of the food itself: **pH, a_x, nutrients, redox/O_2, natural antimicrobials** (lysozyme in egg, eugenol, allicin), physical barriers (rind, shell).

Extrinsic = the storage environment: **temperature** (master variable), **relative humidity** (sets surface a_x), and **gas atmosphere** (O_2/CO_2/N_2). MAP/vacuum suppresses aerobes but can select anaerobes (*Clostridium*) and psychrotrophs.

Trap: know which factor is intrinsic vs extrinsic — temperature & atmosphere are extrinsic; pH & a_x are intrinsic. **Implicit** factors (a third group) cover microbe-microbe interactions: competition, synergy & antagonism between the resident flora — the basis of protective cultures.

12 · Hazards · Types ★ LEC 9

Spoilage organisms reduce quality (odour, gas, slime) but usually aren't dangerous (*Pseudomonas*, yeasts/moulds). **Pathogens** cause disease, often *without* altering smell/look — the danger. **Indicators** signal poor hygiene: coliforms/*E. coli* (faecal), total viable count.

Trap: a food can be **spoiled but safe**, or **unspoiled but dangerous**. An indicator's presence ≠ a pathogen, but raises suspicion.

13 · Infection vs Intoxication ★ GUARANTEED

	INFECTION	INTOXICATION
Cause	live cells multiply in gut	**pre-formed toxin** eaten
Onset	long (12–72 h)	**short (1–6 h)**
Fever	usual	usually none
Cooking	kills → prevents	may NOT help (heat-stable toxin)
Example	*Salmonella*, *Listeria*	*S. aureus*, *C. botulinum*

Toxico-infection = organism ingested, makes toxin *in situ* in the gut (*C. perfringens*, *B. cereus* diarrhoeal, ETEC) — a hybrid: live cells needed, but a toxin does the damage. *B. cereus* uniquely causes both forms: a heat-stable emetic toxin (intoxication, classically reheated rice) and a diarrhoeal toxin made in the gut (toxico-infection).

Trap: intoxication can occur even after reheating kills the microbe, if the toxin is heat-stable. Incubation time is the classic discriminator: minutes-to-hours = pre-formed toxin; many hours-to-days + fever = the organism is multiplying inside you.

13b · Toxins ★ FINAL Q3

Exotoxins — secreted proteins (mostly G+, also G−); potent; usually heat-labile (botulinum/staph exceptions); antigenic → toxoids. Classes:

- **Neurotoxins** — nerves (*C. botulinum*, *C. tetani*)
- **Enterotoxins** — gut (*S. aureus*, *V. cholerae*, ETEC)
- **Cytotoxins** — kill cells (Shiga toxin of O157, diphtheria)

Endotoxins = LPS (lipid A) of the G− outer membrane; structural, heat-stable, released on lysis; fever/septic shock; not toxoids.

13c · Exo vs Endo · Compare ★ TABLE

	EXOTOXIN	ENDOTOXIN
Nature	secreted protein	LPS (lipid A)
Source	G+ & G−, living	G− wall, on lysis
Heat	usually labile	**stable**
Potency	very high	lower
Toxoid?	yes (vaccine)	no

Define toxins first (microbial products that harm the host), then classify exotoxins as neuro-/entero-/cytotoxin with an example each — the full-marks structure for Final Q3.

Two exotoxins break the "heat-labile" rule and matter most in food: **staphylococcal enterotoxin** (survives boiling → vomiting even from reheated food) and **botulinum neurotoxin** (denatures at boiling, but the spore that makes it does not). This is why intoxication can defeat a cook step.

14 · Key Foodborne Pathogens PROFILES

ORGANISM	GRAM · DISEASE TYPE
Salmonella	G− rod · infection · poultry/eggs
Listeria m.	G+ rod · infection · grows at 0–4 °C
E. coli O157	G− rod · Shiga cytotoxin → HUS · low dose
Campylobacter	G− microaerophile · infection · poultry
S. aureus	G+ cocci · intoxication · heat-stable enterotoxin
C. botulinum	G+ spore anaerobe · intoxication · neurotoxin
C. perfringens	G+ spore anaerobe · toxico-infection · warm meats

Trap: *Listeria* at fridge temps defeats "just refrigerate it"; spore-formers survive cooking; O157/*Campylobacter* need a very low infective dose. Match each organism to its control gap: chill fails for *Listeria*, cooking fails for pre-formed staph toxin, and only a full botulinum cook clears *C. botulinum* spores. That organism-specific gap is the heart of the hazard SAQ.

15 · The Swollen-Can Scenario ★★ MTE Q4 / FINAL Q2

Reasoning: a low-acid canned meat with a bad smell + **gas on opening** = under-processing or post-process leakage let spore-forming anaerobes survive/grow. Gas + odour ⇒ gas-producing anaerobes — ***Clostridium*** spp. (incl. the safety-critical ***C. botulinum*** making neurotoxin; or *C. sporogenes* causing putrefactive "swell"). Spores survived an inadequate botulinum cook; the anaerobic interior favours germination → fermentation/putrefaction → gas (CO_2/H_2) + H_2S/amines.

Isolate/identify: aseptic sample → **anaerobic culture** on selective media → **Gram + spore stain** (expect G+ rods with spores) → biochemical (catalase−, anaerobic) → confirm toxin by **ELISA/immunoassay** (or PCR for toxin genes). Prevention = **121 °C / 12-D botulinum cook**.

Trap: don't say "bacteria" — name spore-forming anaerobic *Clostridium* AND explain why.

15b · Spoilage Diagnosis Cues READ THE CAN

SIGN	LIKELY CAUSE
Gas + swell, low-acid	gas-forming anaerobe (*Clostridium*)
Flat sour (no gas)	thermophilic *Bacillus* (flat-sour spoilage)
H_2S blackening	sulfide spoilage (*C. nigrificans*)
Acid + gas, fruit/veg	thermophilic anaerobes

Always pair the *observation* (gas/odour/pH) with the *organism* + its O_2 class and spore status — that is what the SAQ marks.

15c · Indicators & Counts HYGIENE PROXIES

Indicator organisms flag probable contamination rather than being the hazard: **coliforms / *E. coli*** (faecal), Enterobacteriaceae, **total viable / aerobic plate count** (general hygiene), *Enterococcus*. An indicator's presence ≠ a pathogen, but signals poor process control — the basis of the standard plate-count enumeration in the report.

16 · Predictive Microbiology LEC 11 · ILO 5

Quantitative microbiology: capture growth/survival/death responses to environment as **mathematical models**, to *predict* behaviour without testing every product. Three levels:

- **Primary** — microbial number vs *time* at fixed conditions (Gompertz, Baranyi); gives μ, lag, N_{max}.
- **Secondary** — how those params change with *environment* (T, pH, a_x) — square-root/Ratkowsky, Arrhenius.
- **Tertiary** — software combining both (ComBase, Pathogen Modeling Program).

Uses: shelf-life & risk prediction, HACCP critical limits, product design, "what-if" scenarios, fewer challenge tests, objective decisions. **Limits:** valid only within the range used to build them (no extrapolation); assume homogeneous conditions; lab broth ≠ real food matrix/competition; need validation in the actual product. The Ratkowsky square-root model is the classic secondary form linking μ to temperature.

Trap: know primary/secondary/tertiary + one example each; never extrapolate beyond the validated boundaries. Validation checks the model's **bias** (does it over- or under-predict?) and **accuracy** against real observed counts — predictive ≠ a replacement for verification.

17 · Thermal Death Kinetics ★ LEC 12 · D/Z/F

At a lethal temperature, death is **first-order/exponential** — a constant *fraction* dies per unit time, so survivors fall log-linearly. Implication: you reach probabilities, never true zero → "commercial sterility."

VALUE	DEFINITION
D	time at fixed T to kill 90% (1 log)
z	°C change to alter D **10-fold**
F	total equivalent process time (F_0 at 121.1 °C, z=10)

12-D botulinum cook ($F_0 \approx 3$ min) = a 12-log reduction of *C. botulinum* spores for low-acid canned foods.

Trap: D is for ONE temperature; z links D across temperatures; F is total process time. Death is log-linear → sterility is statistical.

17b · Worked · D/z SHOW IT

If $D_{121} = 0.25$ min for *C. botulinum*, a **12-D** cook needs 12 × 0.25 = 3 min at 121 °C (= $F_0 \approx 3$). With z = 10 °C, dropping to 111 °C makes D ten-fold larger (2.5 min) → the same 12-log kill now needs ~30 min.

To cut a population from 10^6 to 10^0 (6 logs) at a D of 1.5 min ⇒ **6 × 1.5 = 9 min**. Process time = (logs to kill) × D. Spores have far higher D than vegetative cells — which is exactly why pasteurisation (a short, mild process) clears the latter but not the former, and why low-acid canning must reach the harsh 121 °C cook.

17c · Logarithmic Death WHY "COMMERCIAL"

Because a constant *fraction* (not number) dies each interval, the survivor curve never hits zero — only smaller probabilities. So canning aims for commercial sterility (a defined low probability of a surviving spore, e.g. 12-D ⇒ a 1-in-10^{12} chance per can), not absolute sterility. This is also why D/z/F feed directly into HACCP critical limits.

18 · Pasteurisation vs Sterilisation ★ LEC 12

Pasteurisation — mild heat killing *vegetative* pathogens & most spoilers + reducing numbers, NOT spores. Milk: LTLT 63 °C/30 min, HTST 72 °C/15 s, UHT 135–150 °C/few s. Targets the most heat-resistant non-spore pathogen.

Sterilisation — destruction of **ALL** microbes incl. spores. **Commercial sterility** = practical absence of organisms able to grow in normal storage (canning, 121 °C); autoclave = 121 °C, 15 psi, 15 min.

Trap: pasteurisation does NOT kill spores or all microbes — **spores & phages survive pasteurisation**. "Commercial sterility" ≠ absolute sterility.

19 · Other Inactivation NON-THERMAL · CHEMICAL

- **Irradiation** (gamma, e-beam, UV) — damages DNA; cold pasteurisation
- **HPP** — high pressure disrupts membranes/proteins, minimal heat damage
- Pulsed electric field, ultrasound, cold plasma — emerging
- **Chemical:** organic acids, **nitrite** (anti-*Clostridium* in cured meat), SO_2, bacteriocins (nisin), smoke
- Drying/salting (↓a_x); **chilling/freezing** = static, not lethal; fermentation

Trap: freezing/chilling & low a_x *inhibit* but don't reliably *kill* (esp. spores); *Listeria* still grows when chilled.

20 · Preservation Logic RECAP

Four levers: **(1) prevent contamination** (hygiene, asepsis); **(2) inhibit growth** (chill, ↓a_x, ↓pH, MAP, preservatives — the hurdle set); **(3) inactivate/kill** (heat, irradiation, HPP); **(4) limit access** (packaging). Choose by target organism, matrix, shelf-life & quality — most real products stack several (hurdle technology) rather than relying on one.

21 · HACCP (Applied Thread) WHERE IT ALL FEEDS

Hazard Analysis and Critical Control Points — a preventive system controlling biological/chemical/physical hazards *through* the process, not by end-product testing. The 7 principles: (1) hazard analysis → (2) determine **CCPs** → (3) set **critical limits** (core ≥ 72 °C, pH ≤ 4.6, $a_x \leq 0.85$) → (4) monitor → (5) corrective action → (6) verification → (7) records. Predictive models & D/z/F values set the critical limits at the CCPs. A CCP is a step where control is essential & can be applied — not every control step is a CCP. HACCP sits on prerequisite GMP/GHP programs and is regulator-required; it controls hazards in-process rather than relying on testing the finished product.

Side 2 · Quick Traps MEMORISE

$N = N_0 \cdot e^{(\mu t)}$ — μ & t dominate over N_0
D = 1-log time · z = °C for 10× D · F = total
pasteurise ≠ sterilise (spores/phages survive)
Infection = live cells · intoxication = pre-toxin
endotoxin = LPS, G-, heat-stable
pH 4.6 = C. botulinum line · danger zone 4–60 °C

SIA → *Name the organism + a number every time, and on Q7 write the logs. Method marks survive a slipped final figure.*