Composite virulence: useful metric or conceptual trap?

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Abstract

Virulence, defined as the harm an infection causes to its host, is central to evolutionary ecology, yet it remains difficult to quantify because infection impact is multidimensional, dynamic, and context-dependent. Composite measures have therefore emerged to summarise multiple traits into a single estimate of infection-induced harm. However, every composite embeds biological assumptions about which traits represent virulence, how they relate to one another, and which components of harm are prioritised. When these assumptions remain implicit, composites can obscure causal mechanisms and compromise interpretation. We argue that composite virulence should be treated as a biologically-grounded measurement strategy rather than a definition of virulence. Used transparently, composite approaches can strengthen evolutionary inference; used carelessly, they risk concealing rather than clarifying infection biology.

Keywords: infection harm, virulence, transmission, composite metric, host-parasite interactions, pathogenicity

1. The composite virulence dilemma

1.1. Why virulence resists simple measurements

Virulence, defined here as *the harm inflicted by infection on the host* [1–3] **(Box 1)**, is central to evolutionary ecology, public health, and livestock management [4–7]. Together with transmission [8,9], it is used to characterize host-parasite interactions [10–14], track evolutionary change [15–18], assess risks from circulating and emerging infections [19–22], guide preparedness and response strategies for future disease outbreaks [23,24], and evaluate interventions designed to mitigate disease severity and spread [25–29].

However, despite its importance, virulence remains difficult to quantify because it is neither a single trait nor a fixed property of a parasite [2,11]. Rather, it emerges from interactions among host genotype, parasite genotype, and environment [2,30]. Infection can reduce host fitness through multiple routes, including delayed development, increased mortality and reduced fecundity (or even sterility) [1]. These fitness consequences are often mediated by physiological or behavioural changes, can emerge at different stages of infection, and frequently depend on ecological context, such that the same parasite can impose very different fitness costs across environments or host populations [2,30]. Consequently, no single measure can capture all biologically relevant components of infection-induced harm. The most informative virulence metric therefore depends on the biological question being asked, the fitness components of interest (e.g., survival, reproduction, or development), and the ecological context in which infection occurs.

This multidimensionality creates a measurement problem. Single proxies are attractive because they seem straightforward, comparable, and often experimentally tractable, yet each capture only one dimension of infection-induced harm. Mortality, for example, directly captures survival [31–33], but it can be insensitive to substantial sublethal costs such as sterilization,

delayed or impaired reproduction [16,34–36]. Likewise, physiological markers such as oxidative stress or inflammation can change markedly during infection without detectable consequences for survival or reproduction, meaning that they do not necessarily reflect host harm unless their fitness consequences are demonstrated [37]. More fundamentally, infection-associated measurements do not all describe the same biological phenomenon. Some quantify *drivers* of harm (e.g., parasite growth and damage mechanisms), others quantify host-state *mediators* (e.g., pathology, inflammation, dysbiosis or sickness behaviours), whereas others quantify fitness *outcomes* (e.g., survival and reproduction). Although, these layers often covary, they do not always do so and should not be treated as interchangeable measures of virulence because each captures a different stage of the infection process.

Virulence is a conceptual construct that must be inferred from measurable traits rather than observed directly [2]. Different measures therefore capture different biological facets of infection and are appropriate for different biological questions. **Intrinsic** [13,29,38], **realized** [39,40], and **proximate virulence** [41,42] emphasize different aspects of infection **(Box 1)**, but each is usually represented by a single proxy and therefore captures only part of infection-induced harm. This raises a central measurement challenge: how should multidimensional harm be represented in a way that remains biologically interpretable for the question at hand?

1.2. Distinct routes to infection-induced harm.

Similar reductions in host survival or reproduction can arise through fundamentally different biological processes. Virulence may increase through greater parasite growth and host resource exploitation, or through differences in per-parasite pathogenicity (PPP), whereby parasites generate more damage at a given parasite burden through cell and tissue injury, inflammatory collateral damage, and toxin- and effector-mediated dysfunction [11,43–46]. More broadly, infection can affect hosts through at least three partially separable processes: (i) host

77 exploitation, whereby parasites divert resources and damage tissues through replication and
78 nutrient sequestration [11,43,47]; (ii) direct injury, in which invasion and growth cause cell
79 lysis, barrier disruption, vascular obstruction, or organ dysfunction even at similar parasite
80 loads [45,46,48–50]; and (iii) host-mediated damage, whereby immune activation generates
81 immunopathology such as oxidative stress or cytokine-driven tissue damage [51,52]. These
82 processes can covary, but they do not always, and they often differ in timing and relative
83 contribution to disease. Consequently, similar measures of virulence may conceal
84 fundamentally different routes to infection-induced harm.

85 Malaria provides a clear example of why these distinctions matter. *Plasmodium spp.* increase
86 harm not only through parasite replication but also through erythrocyte destruction,
87 cytoadherence-mediated microvascular obstruction, and heme-driven oxidative damage, which
88 amplifies inflammation and tissue dysfunction [53,54]. Consequently, two infections can show
89 similar parasite densities yet differ markedly in anaemia, inflammatory pathology, and risk of
90 severe disease because harm reflects different combinations of exploitation, direct injury, and
91 host-mediated damage. Mortality alone collapses these processes into a single endpoint,
92 obscuring both the mechanisms through which parasites inflict harm and the biological
93 processes on which selection ultimately acts [16,55].

94 **1.3. Why composites emerged**

95 The multidimensional nature of infection is not unique to virulence research. Several adjacent
96 disciplines already approach infection effects as interconnected suites of traits rather than
97 isolated endpoints. Chemical ecology, plant defense ecology, and eco-immunology commonly
98 analyse defence chemistry, host condition, pathology, reproduction, and parasite performance
99 using multivariate or pathway-oriented methods instead of reducing them to a single index [56–
100 58]. These fields recognize that different traits describe different components of host-parasite

interactions and therefore provide complementary rather than interchangeable information. Their experience offers a useful precedent for virulence research, where infection-induced harm likewise emerges from multiple interconnected biological processes.

As infection measurements became increasingly multidimensional, several disciplines developed summary metrics to represent disease in a tractable way. In clinical and veterinary medicine, disease severity scores have long combined multiple signs and symptoms into standardized summaries, reflecting the practical reality that disease burden is expressed across several dimensions [16,17,47,59–61]. Plant pathology offers particularly influential precedents because it formalized multidimensional disease impact into quantitative metrics [62–64]. Disease incidence (how many hosts or tissues are infected) and disease severity (how much damage occurs per infected unit) are routinely analysed together because they capture distinct dimensions of disease [63,65]. Likewise, the area under the disease progress curve (AUDPC/AUC) integrates repeated measurements of disease severity over time into a single estimate of cumulative burden [65–67]. Collectively, these approaches illustrate two general principles: infection impact is inherently multidimensional and reducing it to a single summary measure inevitably involves a trade-off between simplicity and biological information.

Evolutionary ecology reached similar conclusions, but from a different perspective. Rather than summarizing disease severity alone, multivariate approaches were adopted to understand how parasite traits translate into fitness costs and evolutionary change. In plant–virus systems, for instance, tolerance is quantified by tracking infection-induced changes across multiple life-history traits, revealing that genotypes can differ in how infection costs are distributed across growth and reproduction rather than in total damage alone [68,69]. Similar logic motivates the routine use of trait panels in animal and microbial systems, where infection effects on survival and reproduction are analyzed alongside host-state mediators (e.g., pathology, oxidative stress, behaviour) and parasite traits (e.g., burden, growth, persistence) [16,43,55,70,71]. The

rationale is straightforward: mortality is a downstream outcome of infection, whereas selection acts on parasite traits through their effects on host survival, reproduction, and transmission opportunity. Multivariate measurement therefore follows directly from the biology, providing a natural way to capture infection trajectories, trade-offs, and context-dependence.

As multivariate trait panels became increasingly common, researchers naturally sought ways to summarize this complexity into a single measure. Composite indices offered an attractive compromise by providing concise, comparable representations of infection while retaining information from multiple traits. However, this simplification is never biologically neutral. Every composite requires decisions about which dimensions of harm to include, how they should be combined, and what the resulting measure is intended to represent. These decisions are often appropriate and unavoidable, but they also shape the biological meaning of the composite itself **(Fig. 1)**.

Every composite requires decisions about trait selection, weighting, independence, and biological target. Once compressed into a single value, these choices become largely invisible, yet they determine how virulence is interpreted. Consequently, two studies analyzing the same infection system could reach different conclusions simply because they constructed different composites.

1.4. Composites clarify, but can also conceal

Crucially, these assumptions extend to time. The biological consequences of the same trait can differ markedly depending on when it is expressed or measured during infection, from negligible effects to substantial fitness cost, because virulence and transmission emerge from dynamic infection trajectories rather than static measurements [72,73]. Early physiological or behavioural changes may be compensated or reversed, but they can also trigger cascading effects that shape later survival, reproduction, or transmission. Conversely, similar changes

150 expressed later in infection may have major immediate consequences or relatively little impact
151 if key fitness outcomes have already been determined, such that parasite density measured at
152 different stages of infection can carry very different evolutionary significance [74].
153 Consequently, the biological significance of a trait cannot be separated from its timing.
154 Composite measures should therefore either integrate information across infection or explicitly
155 define the phase they represent. Similar temporal dependencies have been documented across
156 diverse host–parasite systems, including *Ophryocystis elektroscirrha* infections of monarch
157 butterflies, where the timing of parasite development strongly influences both host fitness and
158 transmission potential [55,75].

159 Composite measures nevertheless offer clear practical advantages by summarizing
160 multidimensional infection outcomes and facilitating comparisons among parasite genotypes,
161 host genotypes, treatments, or environments [76,77]. However, every composite also embeds
162 biological decisions about which traits define infection-induced harm, how they are combined,
163 and which dimensions of infection are emphasized or ignored [78,79]. The framework below
164 is intended to make those biological decisions explicit and therefore open to evaluation and
165 refinement **(Box 2; Fig. 2)**. Although presented primarily as a prospective workflow for
166 experimental design **(Fig. 2a)**, the same principles can also be applied retrospectively to
167 existing multidimensional datasets **(Fig. 2b)**.

168 **2. A framework for biologically-grounded composite virulence**

169 Composite metrics are motivated by a biological reality: infection affects multiple traits in a
170 dynamic, context-dependent manner. Rather than combining whatever traits happen to be
171 available, composites should be constructed top-down by first defining the biological target
172 and then selecting non-redundant traits that directly represent it. Accordingly, we treat
173 **composite virulence** as a measurement strategy: *a pre-specified multidimensional and defined*

174 *facet of infection-induced harm built to answer a specific biological question*. The framework
175 below is intended to make those biological decisions explicit and therefore open to evaluation
176 and refinement **(Box 2, Fig. 2)**.

177 **2.1. Define the biological target**

178 The first step in constructing an interpretable composite is to define its biological target **(Fig.**
179 **2)**. Different biological questions require different measures of infection impact, and no single
180 composite can represent all facets of virulence simultaneously. In evolutionary ecology, the
181 biological target is often host fitness because selection acts through survival and reproduction
182 [80]. In other contexts, the target may instead be disease burden, mechanistic severity, or
183 population-level impact. The intended biological target therefore determines which traits
184 belong within the composite and which should instead be analysed separately to explain
185 variation in the composite [81].

186 Once the biological target has been defined, candidate traits should be organized according to
187 their role in the infection process **(Fig. 1a; Fig. 2)**. Parasite burden, replication rate, or
188 environmental persistence typically function as drivers of harm. Pathology, immune activation,
189 physiological disruption, and dysbiosis often act as host-state mediators that transmit or modify
190 damage [82]. Survival, reproduction, and related life-history traits represent downstream
191 fitness outcomes through which infection costs ultimately manifest [80]. Although all these
192 traits are biologically informative, they occupy different positions within the causal pathway
193 and therefore answer different biological questions.

194 Combining traits across these causal layers can obscure biological interpretation because
195 mechanistic traits become embedded within the outcomes they are intended to explain. A
196 practical solution is therefore to construct each composite around a single biological target and
197 to include only traits that directly represent that target while analysing traits from other causal

levels as explanatory variables rather than components of the composite.

2.2. Minimize redundancy

Once traits have been selected from the appropriate causal level, the next consideration is whether each contributes unique biological information **(Fig. 2)**. Composite measures become biased when multiple traits quantify the same underlying process, effectively increasing its influence without biological justification [78]. For example, parasite burden measured using quantitative PCR, microscopy, and colony counts (CFUs) may represent different assays but often capture the same biological phenomenon. Likewise, several inflammatory markers may reflect a common immune response rather than independent dimensions of infection. Such redundancy can unintentionally weight one component of infection more heavily than others, altering the biological interpretation of the composite [83].

Even when the biological target is well-defined, composites can exaggerate differences by treating correlated measures as independent evidence of harm. Multiple symptoms and physiological readouts often reflect shared sickness processes, whereas life-history traits may covary through common physiological or ecological constraints. Additive indices built from overlapping readouts can therefore overemphasize particular dimensions of infection impact by effectively counting the same biological process multiple times [83] **(Fig. 1b)**. Data-driven reductions can also mislead when the dominant axis reflects whichever trait varies most, rather than whichever trait best represents the chosen biological target [79].

Group related measurements into biologically meaningful categories before constructing the composite. Each category should summarize overlapping traits, after which categories can be evaluated for unique contributions to the biological target [78,79].

The consequences of redundancy become apparent whenever different dimensions of infection contribute unequally to the biological target. In parasites that castrate their hosts, for example,

profound reproductive costs can occur with little effect on survival [34,35]. Likewise, in a natural mosquito–microsporidian system, parasites selected for late transmission produced different virulence rankings depending on whether virulence was inferred from mortality or fecundity [16]. These examples illustrate a general principle: composite measures that disproportionately represent one route to infection-induced harm can underestimate others that are biologically more relevant. Composite indices should therefore be accompanied by robustness analyses demonstrating that conclusions are not artefacts of trait selection, scaling, or weighting, particularly when component traits are correlated [61].

2.3. Preserve infection dynamics

Infection is a dynamic process rather than a static event **(Fig. 1c)**. Accordingly, infection dynamics should be explicitly incorporated into composite construction **(Fig. 2)**. However, composite measures are often derived from single time points or endpoints, implicitly assuming that infection-induced harm is constant through time. In reality, parasite growth, host responses, and damage profiles shift throughout infection, often nonlinearly [43,84]. Hosts and parasites can therefore differ not only in the magnitude of infection but also in the timing and sequence of infection-related effects [85,86]. Consequently, two infections may produce similar endpoint pathology or mortality however differ substantially in both host and parasite fitness because costs and transmission opportunities are distributed differently through time or because hosts partially recover [70].

Dynamics also shape the mechanistic interpretation. The same host-state mediator may be protective early in infection yet damaging later, and traits that appear minor at one time point can become decisive when evaluated over the life-history window that matters most. Conversely, severe end-stage pathology may have limited evolutionary consequences if it occurs after most reproduction or parasite transmission has already taken place [72,74].

Collapsing time can therefore erase precisely the variation that distinguishes parasite strategies, and the selective pressures acting on both.

Time should be treated as part of the phenotype rather than a nuisance. Whenever possible, longitudinal measurements should be retained because they preserve information about the order, duration, and timing of infection effects. When some degree of summarisation is necessary, the chosen metric should match the biological question being addressed. Survival curves, age-specific fecundity schedules, or repeated performance measures can sometimes be reduced to cumulative reproduction, hazards within a defined biologically relevant window, or AUC-style measures, but only when these summaries preserve the aspect of infection dynamics relevant to the hypothesis under study. When full longitudinal tracking is infeasible, a window-based approach often captures much of the relevant information: define biologically justified phases of infection (e.g., establishment, peak, late-stage, recovery) provide a useful compromise. Reporting timing features such as onset, peak, recovery, or time-to-threshold alongside magnitude often captures biologically important differences that would otherwise be lost.

The guiding principle is alignment. The time window used to construct a composite should correspond to the period that matters for the host life-history and the parasite transmission. Without this alignment, "overall virulence" becomes partly a property of the sampling design rather than of the infection process itself.

2.4. Account for ecological context

Composite virulence measures are frequently estimated under a single set of laboratory conditions and then treated as properties of a particular host–parasite interaction **(Fig. 2)**. However, virulence is inherently context-dependent because infection-induced harm emerges from interactions among host and parasite genotypes, and their shared environment (*i.e.*,

genotype x genotype x environment) **(Fig. 1d)** [2,87–89]. Host genotype, age, nutrition, temperature, microbiome composition, density, co-infection, and exposure route can each alter not only the magnitude of infection-induced harm, but also which route to harm dominates, or even whether a given trait has detectable fitness consequences. Consequently, a composite constructed under one ecological context may not accurately represent infection impact under another.

Ecological context often acts on specific stages of the infection process rather than on virulence as a whole. Temperature may amplify parasite replication (drivers of harm), nutrition may alter tolerance and repair capacity (host-state mediators), microbiome composition may modify immune activation and dysbiosis (host-state mediators), and exposure route may change which host behaviours determine transmission opportunity (life-history outcomes relevant to epidemiological impact) [30,88,90]. Analytical approaches that explicitly represent these causal pathways, such as path analysis or structured equation modelling [91–93], can distinguish where ecological context acts within the infection process rather than simply whether it changes the final composite. Under this view, context-dependence is not random variation around a single virulence value, but structured variation in how infection translates into fitness costs across ecological conditions.

Where feasible, the most informative representation of virulence is a reaction norm rather than a single number because it makes context-dependence explicit [94]. Rather than relying on a single composite value, composites (or their component categories) can be evaluated across ecologically meaningful contexts, allowing comparisons of both average infection impact and its sensitivity to environmental change. Even modest factorial designs can reveal whether differences are robust (parallel reaction norms) or context-dependent (crossing reaction norms), and whether variation arises primarily from host, parasite, or environmental effects, or from their interactions. When fully factorial experiments are impractical, measuring key

contextual variables and incorporating them explicitly into statistical models is often preferable to averaging across them, which risks obscuring biologically meaningful variation.

The guiding principle is context-dependence. Composite virulence should be interpreted as a property of a host-parasite interaction under defined ecological conditions rather than as an intrinsic property of either partner. Measuring composites across relevant environmental gradients helps distinguish robust patterns from context-specific responses and makes explicit the ecological domain over which biological inference is valid.

3. Concluding Remarks

Virulence research is entering an era in which multidimensional phenotyping is becoming routine. Advances in automated imaging, behavioural tracking, omics technologies, and longitudinal monitoring increasingly allow infection to be quantified across multiple biological scales simultaneously. As these datasets grow in complexity, the challenge is no longer whether infection-induced harm should be represented by one trait or many, but how these measurements can be translated into biologically meaningful estimates of virulence. Composite approaches are likely to become increasingly valuable, provided their construction follows biological rather than analytical logic. Defining the biological target, minimizing redundancy, preserving infection dynamics, and accounting for ecological context offer a practical framework for making the assumptions underlying composite virulence explicit, interpretable, and testable.

Many important challenges nevertheless remain **(see Outstanding Questions)**. Can different dimensions of infection-induced harm be integrated without obscuring causal mechanisms? Which aspects of virulence are under direct selection, and which simply reflect correlated consequences of infection? Under what ecological conditions do parasite strategies remain robust, and when do they fundamentally change? Addressing these questions will require closer

integration between evolutionary ecology, epidemiology, physiology, quantitative biology, and increasingly, data science. Ultimately, the value of any composite measure will never lie in the number of traits it combines, but in how transparently those traits capture the biology of infection-induced harm.

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Author contributions

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Data availability

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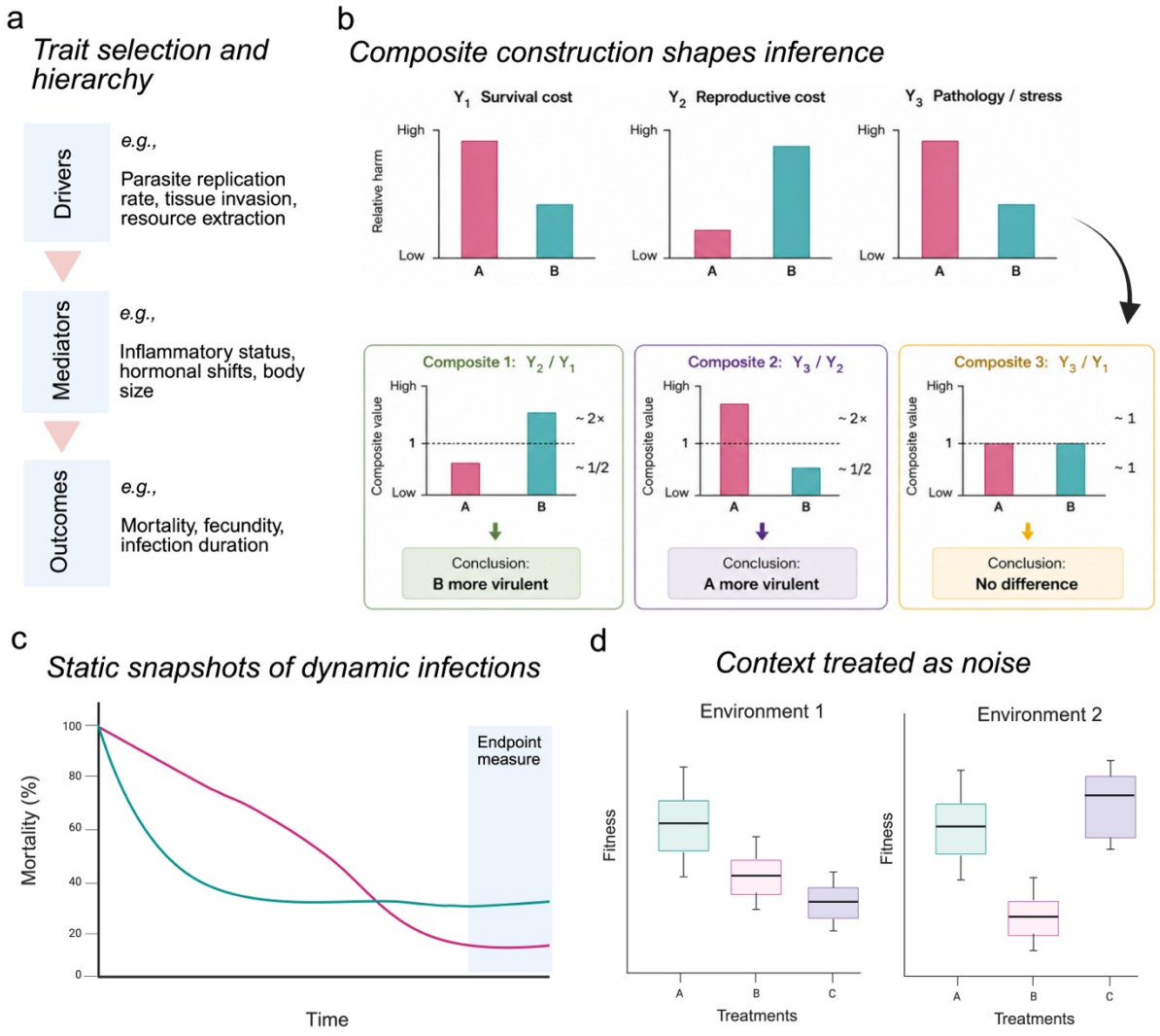

Figure 1. Common pitfalls of composite virulence metrics. (a) Trait selection and hierarchy. Composites often combine variables that operate at different biological levels: drivers (e.g., replication rate, tissue invasion, resource extraction), mediators (e.g., inflammatory status, hormonal shifts, body size), and outcomes (e.g., mortality, fecundity, infection duration). Mixing these levels obscures causal structure and conflates mechanisms with their downstream consequences. **(b)** Composite construction shapes inference. The same set of underlying traits can generate contrasting conclusions depending on how they are combined. In this hypothetical example, treatments differ across survival, reproduction, and physiological stress, but alternative composites constructed from different combinations of these traits produce different

rankings of virulence. Because composites implicitly determine which traits are emphasized, reduced, or balanced against one another, inference can depend as much on composite construction as on the underlying data. **(c)** Static snapshots of dynamic infections. Virulence unfolds over time, yet composite measures often rely on endpoint values. Distinct infection trajectories can yield similar final values while differing in timing, peak severity, or recovery dynamics, obscuring biologically relevant variation. **(d)** Context treated as noise. Environmental conditions reshape trait expression and treatment rankings. When ecological context is ignored or averaged across, biologically meaningful genotype × genotype × environment interactions are interpreted as residual variation rather than determinants of virulence expression. Together, these pitfalls illustrate how composite virulence indices can generate simple numerical summaries while concealing causal structure, temporal dynamics, ecological contingency, and the biological assumptions embedded in composite construction. Created in BioRender. lab, K. (2026) https://BioRender.com/u8u0z52.

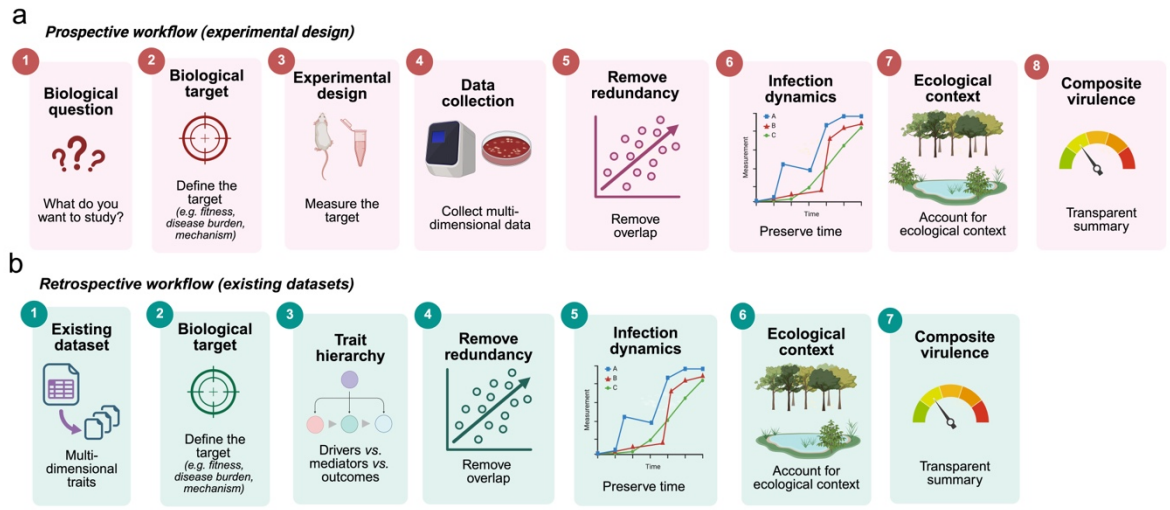

Figure 2. Constructing biologically-grounded composite virulence. (a) In prospective studies, composite virulence should be considered during experimental design. A biological question defines a single biological target, which determines trait selection before data

collection. The resulting measurements are then evaluated for redundancy, infection dynamics, and ecological context before being integrated into a transparent composite. **(b)** Existing multidimensional datasets can be analysed using the same framework by defining the biological target first, organizing traits according to their causal role, and then evaluating redundancy, temporal dynamics, and ecological context. In both cases, composite virulence is treated as a biologically grounded measurement strategy whose interpretation depends on explicit biological assumptions rather than statistical summarization alone. Created in BioRender. lab, K. (2026) https://BioRender.com/d9g28zw.

Box 1. Glossary

Virulence: Infection-induced harm to the host. In evolutionary contexts, virulence is often inferred from effects on host fitness-related traits such as survival, reproduction and development.

Intrinsic virulence: Harm caused by a parasite genotype when measured under standardized host and environment conditions, typically for comparative purposes.

Realized virulence: The total impact of infection expressed in a particular ecological and evolutionary context, incorporating variation among hosts, parasites and environments.

Proximate virulence: Mechanistic processes and host-state changes associated with infection (*e.g.,* pathology, inflammation, tissue damage, dysbiosis) that contribute to harm but are not equivalent to fitness consequences unless their effects on survival, reproduction or development are demonstrated.

Composite virulence: A measurement strategy that integrates multiple traits into a single representation of infection impact for a specified biological target or question.

Biological target: The specific facet of infection impact that a measurement is intended to represent (*e.g.,* life-history costs, disease burden, mechanistic severity, or epidemiological impact).

Multivariate measurement: The simultaneous assessment of multiple traits to characterize different dimensions of infection impact without necessarily reducing them to a single summary value.

Drivers of harm: Parasite traits or processes that generate physiological stress or damage such as replication rate, parasite burden, persistence, or resource exploitation.

Host-state mediators: Host responses or conditions that transmit, amplify or mitigate infection-induced damage, including immune activation, pathology, physiological disruption, oxidative stress and dysbiosis.

Life-history outcomes: Effects of infection on host survival, reproduction, development or other fitness-related traits that determine evolutionary consequences.

Trait hierarchy: The organization of infection-related traits according to their causal role in the infection process, distinguishing drivers, host-state mediators, and life-history outcomes.

Host exploitation: The component of infection-induced harm resulting in parasite growth-and resource use, often reflected in the diversion of host resources, energetic costs, or tissue damage associated with replication.

Per-parasite pathogenicity (PPP): Harm caused per unit of parasite burden, reflecting mechanisms independent of parasite density, such as toxin production, inflammation, immune-mediated pathology, or tissue-specific damage.

Reaction norm: The pattern of phenotypic expression of a trait across environmental conditions. In the context of virulence, reaction norms describe how infection impact changes across ecological contexts.

Context-dependence: Variation in infection impact arising from host genotype, parasite genotype, environmental conditions, or their interactions, such that the magnitude or dominant route to harm changes across contexts.

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Box 2. A practical framework for constructing biologically-grounded composite virulence

Composite virulence is best treated as a measurement strategy rather than a definition. Its interpretability depends on making a small set of biological decisions explicit.

0) Decide whether a composite is necessary: Not all multidimensional datasets require a single summary metric. When relationships among traits, causal pathways, timing, or context-dependence are central to the question, a multivariate representation may be more informative than a composite.

1) Define the biological question: State which facet of infection impact the composite is intended to represent (e.g., life-history costs, disease burden, mechanistic severity, or epidemiological impact) and which biological question it is designed to answer. Build each composite around a single biological target and select only traits that directly represent that target. Organize candidate traits according to their causal role (drivers of harm, host-state mediators, or life-history outcomes) and analyse traits from other causal levels separately as explanatory variables rather than incorporating them into the composite.

2) Minimize redundancy: Avoid including multiple measurements that capture the same biological process. Group correlated traits into biologically meaningful categories before constructing the composite, and evaluate whether each category contributes unique information to the biological target. Report robustness analyses showing that conclusions are not driven by weighting, scaling, or correlated measurements.

3) Preserve infection dynamics: Treat time as part of the phenotype. Whenever possible, retain longitudinal measurements or summarize infection using biologically justified windows or timing metrics (e.g., onset, peak, recovery). The temporal scale of the composite should match the biological process under investigation.

4) Account for ecological context: Interpret composite virulence relative to the ecological conditions under which it was measured. Whenever feasible, evaluate composites across biologically relevant environmental gradients, compare reaction norms rather than single-condition rankings, and incorporate contextual variables explicitly into statistical models rather than averaging across them.

5) Retain transparency: Report component traits or category-level summaries alongside any single-number index whenever possible. Clearly describe trait selection, scaling, weighting, and any dimensionality reduction so that the biological assumptions underlying the composite remain explicit and reproducible.

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