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Scheduling Physical Activity in Higher Education: The Unmeasured Time-Course of Acute Exercise Effects on Students' Executive Function

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Abstract

Introduction: *Universities schedule physical activity. Compulsory physical education credits, campus recreation hours and activity breaks within long lectures are timetabling decisions, and each is increasingly justified by evidence that a single bout of exercise improves executive function. Each justification depends on a quantity the literature seldom reports: how long the improvement lasts. This review asks why the persistence of the acute effect has remained unspecified, and what follows for scheduling and for research design.*

Methodology: *This is a critical narrative review. We assembled a purposive collection of meta-analyses, experimental studies in young adults and mechanistic reviews concerning acute exercise and cognition, together with physiological literature on the time courses of the mechanisms most often invoked. Sources illustrate design decisions rather than forming a representative sample; no pooled estimate is produced.*

Themes and findings: *Seven themes are developed. The field reports a point estimate where a decay function is required, because cognition is typically assessed once, at a delay that is variable and frequently unstated. The candidate mechanisms — cortical arousal, cerebral haemodynamics and neurotrophic signalling — operate on time constants differing by an order of magnitude, and so predict incompatible decay curves that a single measurement cannot distinguish. Intensity and post-exercise delay are structurally confounded, which may account for the unstable intensity moderator across meta-analyses. Students who benefit most may not be those who benefit longest. The question universities face is a timetabling question that current evidence cannot answer. Unmodelled delay heterogeneity plausibly contributes to the heterogeneity routinely reported. And acute persistence is routinely conflated with chronic adaptation.*

Conclusion and recommendations: *We propose a ten-item reporting minimum centred on exact time from exercise cessation to task onset, and argue for multi-timepoint designs that estimate a decay curve rather than a single contrast.*

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1. Introduction

Higher education institutions make decisions about when students move. Many national systems require physical education credits of undergraduates: China's universities administer compulsory physical education across the first two years, Malaysian institutions carry co-curricular requirements, and campus recreation provision is a standard component of student services elsewhere. Alongside these formal requirements, universities decide when gymnasia open, whether activity breaks are built into three-hour teaching blocks, and where sport facilities sit relative to the academic timetable.

Increasingly, these decisions are justified on cognitive grounds. The proposition that a single bout of physical activity temporarily improves cognitive control is among the more secure findings in exercise psychology. It has survived meta-analysis across the general literature (Chang et al., 2012; Lambourne & Tomporowski, 2010), in analyses distinguishing intensity bands (McMorris & Hale, 2012), and in syntheses spanning age and fitness groups (Ludyga et al., 2016; Verburgh et al., 2014). It has been demonstrated behaviourally in undergraduate samples (Pontifex et al., 2009; Sibley & Beilock, 2007) and corroborated by prefrontal imaging during post-exercise cognitive tasks in young adults (Byun et al., 2014; Yanagisawa et al., 2010). Few relationships in the field rest on such convergent evidence.

The institutional inference contains a hidden quantity. If a benefit is to be exploited by the lecture, seminar or private study that follows, the benefit must still be present when that activity begins. A ten-minute effect and a ninety-minute effect authorise entirely different timetables. The first supports an activity break immediately before a demanding class and nothing more; the second supports scheduling physical education in the morning and expecting returns across the teaching day. University administrators have read the literature as though it settled this question. It has not addressed it.

The reason is a design convention rather than an oversight of interpretation. The overwhelming majority of acute-exercise experiments assess cognition once after the bout. That single assessment is placed at a delay that varies substantially across studies — from immediately upon cessation to an hour or more — and that is often described only loosely, if at all. Under this convention, each study yields one number: the difference between exercise and control at one unspecified point on a curve whose shape nobody has measured. Aggregating such numbers produces an average of samples taken at different, unrecorded positions along an unknown function.

It is worth being precise about what is and is not being claimed. The claim is not that researchers have been careless. Fixing a single assessment point is entirely reasonable when the goal is to establish that an effect exists, and the convention served the field well throughout the period in which that goal was the priority. Nor is the claim that persistence has never been examined; several studies have deliberately imposed a delay, and at least one methodological

treatment has addressed timing directly. The claim is narrower and, we think, harder to dismiss: persistence has never been treated as the estimand — as the quantity a study sets out to measure — and in consequence the literature contains no characterisation of how the effect changes with time since cessation, for any population or any protocol. A quantity on which every scheduling application depends has been left to inference from studies that were not designed to supply it.

This is not a minor methodological complaint, because the mechanisms proposed to explain the effect do not share a time course. Cortical arousal mediated by catecholaminergic activity dissipates within minutes of cessation. Exercise-induced changes in cerebral blood flow normalise over a similar or somewhat longer interval. Circulating brain-derived neurotrophic factor peaks at or near the end of exercise and declines over tens of minutes. Affective and motivational changes may persist considerably longer. These are not competing accounts of the same curve; they are accounts of different curves. A field that measures at one point cannot adjudicate between them, and a field that does not record which point it measured cannot even accumulate the evidence that would.

The purpose of this review is therefore threefold. First, to establish that the persistence of the acute effect is a distinct and currently unanswered question, rather than a detail subsumed under existing findings. Second, to show that the temporal structure of the candidate mechanisms makes this question theoretically consequential and not merely applied. Third, to specify what would have to change in reporting and design for the question to become answerable, and to argue that these changes are inexpensive relative to what they would deliver.

We restrict attention to university students and comparable young-adult samples. This is the population in which the acute effect has been most extensively characterised, it is the population whose timetables higher education institutions actually control, and it avoids the developmental confounds that make extrapolation across age unsafe (Diamond, 2013). We consider executive function rather than cognition in general, since it is the domain in which the acute effect is most consistently observed and the domain to which academic applications appeal.

2. Literature review

2.1 The acute effect and its established moderators

Executive function is conventionally decomposed into inhibition, updating of working memory and cognitive flexibility — components that are correlated but separable, and whose separability was established in precisely the undergraduate populations at issue here (Miyake et al., 2000; Diamond, 2013). The acute exercise literature has concentrated on inhibitory control, most often assessed with flanker or Stroop paradigms, and on working memory.

Meta-analytic estimates place the acute effect in the small-to-moderate range. Etnier et al. (1997) provided the first large synthesis of fitness and exercise effects on cognition; Chang et al. (2012) synthesised the acute literature specifically and reported effects that varied

systematically with exercise intensity, duration and the timing of assessment relative to the bout. Verburgh et al. (2014) and Ludyga et al. (2016) reached comparable conclusions across age bands including young adults, with Ludyga et al. reporting moderation by age and fitness. Moreau and Chou (2019) examined high-intensity protocols specifically and cautioned that the evidence base is smaller and more heterogeneous than the enthusiasm surrounding it. An individual-participant-data synthesis by Ishihara et al. (2021) offered the most granular treatment to date and again identified timing among the factors requiring attention.

Two moderators recur. The first is intensity, usually discussed through an inverted-U framing that descends from Yerkes and Dodson (1908) by way of arousal theory: moderate intensities are held to optimise performance while very low and very high intensities do not. Chang et al. (2015) added a duration dimension, reporting a dose–response relation between bout length and subsequent cognition in young adults. The second moderator is the individual's baseline standing. Sibley and Beilock (2007) found that the working-memory benefit of an acute bout in college students depended on individual differences in baseline capacity, with the largest gains among those who began lower — a pattern consistent with a compensatory rather than a uniform mechanism.

A third source of variation deserves note, because it interacts with everything that follows. The acute effect is not uniform across executive function components or across the tasks used to index them. Inhibitory control assessed by interference paradigms has been examined far more often than updating or flexibility, and the tasks themselves differ in duration, in the number of trials required for a stable estimate, and in their susceptibility to practice effects. This matters temporally as well as psychometrically. A task requiring several minutes of testing does not sample a single moment; it integrates performance across a window. Where that window sits relative to exercise cessation is determined by the position of the task within the battery, so task selection and assessment timing are not independent design decisions — although they are almost always reported as though they were.

2.2 The mechanisms invoked

Several mechanisms are routinely proposed. The reticular-activating hypofrontality model holds that exercise transiently reallocates neural resources, with consequences for prefrontal function that depend on intensity and duration (Dietrich & Audiffren, 2011). Arousal accounts emphasise catecholaminergic modulation of cortical excitability and its influence on attentional processing (McMorris & Hale, 2012; Lambourne & Tomporowski, 2010). Haemodynamic accounts point to exercise-induced increases in cerebral perfusion and their regulation (Ogoh & Ainslie, 2009), and near-infrared spectroscopy studies in young adults have linked post-exercise Stroop performance to increased dorsolateral prefrontal activation (Byun et al., 2014; Yanagisawa et al., 2010). Neurotrophic accounts implicate brain-derived neurotrophic factor, whose serum concentration rises with acute exercise and has been proposed as a route to enhanced cognitive performance (Piepmeier & Etnier, 2015; Cotman et al., 2007; Ferris et al., 2007). Broader syntheses treat these as complementary rather than mutually exclusive (Basso & Suzuki, 2016; Hillman et al., 2008).

What is rarely made explicit is that each of these mechanisms carries an implicit temporal signature, and that the signatures differ substantially. We return to this in Section 4.2.

2.3 What is already known about persistence

The question is not entirely unexamined. Hogan et al. (2013) assessed affect and cognition following an acute bout in younger and older adults and reported benefits that were not confined to the moment of cessation. Tsukamoto et al. (2016) compared high-intensity interval and moderate-intensity continuous protocols in young adults with explicit attention to post-exercise executive function, and is among the few studies to treat the post-exercise interval as an object of interest rather than a nuisance. Pontifex et al. (2019) provided a methodological treatment of the after-effects of acute activity on cognition that addresses timing among other design considerations, and represents the most direct engagement with the issue we are aware of.

These contributions are valuable but sparse, and they have not been consolidated. There is, to our knowledge, no systematic characterisation of how the magnitude of the acute effect on executive function changes as a function of time since exercise cessation — no estimated decay curve, and no meta-regression treating delay as a continuous moderator. The literature contains the raw material for such an analysis only to the extent that individual studies have reported their timing precisely, which many have not.

3. Methodology

3.1 Type of review

This is a critical narrative review rather than a systematic review. Its object is a design convention and its consequences, not an effect size. A systematic search would be the appropriate instrument for estimating the acute effect; it is not the appropriate instrument for arguing that the field has been estimating the wrong quantity. Accordingly we did not register a protocol, did not conduct an exhaustive search, and produce no pooled estimate.

3.2 Sources and selection logic

We assembled a purposive collection of sources of three kinds. The first comprises meta-analyses and quantitative syntheses of acute exercise effects on cognition, selected to span the period over which the finding consolidated. The second comprises individual experimental studies in university students and comparable young-adult samples, selected because they illustrate a specific design decision about the timing of post-exercise assessment. The third comprises mechanistic and physiological reviews, selected because they permit an estimate of the time constant associated with each proposed mechanism.

Studies are cited as instances, not as a sample. Where we describe a study's design, that description is taken from the study's own reporting. Where a temporal detail could not be established from the primary source, we say so rather than infer it. This constraint has shaped the argument: several claims below are stated qualitatively where a quantitative statement

would have been more persuasive but would have required attributing to studies a precision they do not report. That gap is itself part of what this review is about.

3.3 Limitations of the approach

Three limitations follow directly. Purposive selection cannot exclude the possibility that studies with careful temporal reporting exist and were not located; if so, the argument that reporting is inadequate would need to be softened, though the argument that the evidence has not been consolidated would stand. Single-reviewer selection introduces the usual risk of unrecognised bias. And a narrative review cannot quantify how much of the observed heterogeneity in this literature is attributable to delay; it can only argue that the quantity is currently unestimated and identify what would be required to estimate it.

4. Themes and findings

4.1 A point estimate where a function is required

Consider what a conventional acute-exercise experiment produces. Students are randomised to a bout of exercise or to a control condition; after the bout, executive function is assessed; the group difference is expressed as an effect size. The design yields a scalar.

But the object of inference is not a scalar. If exercise perturbs a physiological system that then returns towards baseline, the effect on cognition is a function of time since cessation — rising, peaking and decaying over some interval. The experiment samples that function once. Which value it returns depends on where the sample was taken.

The consequence is that two competently conducted studies of the same protocol in the same population can disagree entirely for reasons that have nothing to do with sampling error. One testing at three minutes and one testing at forty may be measuring opposite phases of the same curve. Neither is wrong; they are answering different questions, and neither reports which question it answered in a form that permits the other to be located relative to it.

This would be tolerable if delay were incidental. The remainder of this section argues that it is not.

4.2 Mechanisms with mismatched time constants

The mechanisms proposed for the acute effect differ in how quickly they resolve after exercise stops. Table 1 sets out an approximate ordering. The entries are deliberately coarse: they are intended to establish that the mechanisms occupy different temporal bands, not to fix precise durations, which vary with intensity, duration and fitness.

Table 1: Candidate mechanisms for the acute effect and their approximate temporal bands

Mechanism	Direction of change during exercise	Approximate band over which it resolves after cessation	Predicted shape of the cognitive effect
Catecholaminergic arousal and cortical excitability	Increased	Minutes	Largest immediately; rapid decay
Cerebral perfusion and prefrontal haemodynamics	Increased, intensity-dependent	Minutes to tens of minutes	Early peak; moderate decay
Circulating neurotrophic factors	Increased, peaking near cessation	Tens of minutes	Later peak; slower decay
Transient fatigue and peripheral load	Increased	Minutes to tens of minutes	Suppression close to cessation; benefit emerges on recovery
Affect, mood and motivational state	Typically improved	Tens of minutes to hours	Slow decay; least intensity-dependent

Source: Developed for this research, drawing on Basso and Suzuki (2016), Byun et al. (2014), Cotman et al. (2007), Dietrich and Audiffren (2011), Ogoh and Ainslie (2009) and Piepmeier and Etnier (2015). Bands are indicative orderings, not established durations.

The point of the table is the spread. If the effect is carried principally by cortical arousal, it should be largest immediately after cessation and substantially gone within a short interval. If it is carried by neurotrophic signalling, the peak should be later and the decay slower. If affective or motivational change is doing the work, persistence could extend well beyond either. These are empirically distinguishable predictions, and distinguishing them is precisely what a single post-exercise measurement cannot do.

The theoretical cost is considerable. Mechanistic debate in this literature proceeds largely by correlating a biomarker or an imaging index with a cognitive score at one time point. But co-occurrence at one moment is weak evidence for mediation when several candidate processes are elevated simultaneously. The shape of the decay is a far more discriminating signature than the height of a single point, and it is available at modest cost: it requires only that cognition be measured more than once.

4.3 The intensity–delay confound

Exercise intensity and post-exercise delay are not independent in practice. Higher intensities produce greater peripheral fatigue and larger transient physiological perturbation. A cognitive

task administered immediately after a vigorous bout is therefore administered under conditions — elevated heart rate, altered respiration, subjective fatigue — that may themselves depress performance, independently of any effect on the underlying cognitive process. The same bout assessed twenty minutes later, once those conditions have resolved, may show benefit. Tsukamoto et al. (2016) is instructive here precisely because it compared protocols differing in intensity while attending to the post-exercise interval.

This has an important implication for the inverted-U account. The received interpretation is that very high intensities are less beneficial than moderate ones because arousal has passed its optimum. An alternative interpretation is available: high-intensity protocols may be disproportionately assessed close to cessation, and their apparent disadvantage may partly reflect measurement during the recovery window rather than a genuine ceiling on benefit. Kamijo et al. (2007) demonstrated that the interaction between exercise intensity and cognitive task demand is not simple, and the tools exist to separate these accounts. They have not been systematically applied.

We do not claim that the inverted-U account is wrong. We claim that as long as intensity and delay covary and delay goes unmodelled, the two accounts are not distinguished by the available evidence, and the instability of the intensity moderator across meta-analyses is consistent with a confound of exactly this kind.

4.4 The window may not belong to every student equally

That individuals with weaker baseline executive function show larger acute benefits is among the more practically important findings in this literature (Sibley & Beilock, 2007), because it identifies the students for whom an intervention would be most valuable. It also raises a question that has not been asked: is the benefit for those students also longer-lasting?

The two properties are logically independent. A larger peak with a faster decay would mean that the students who benefit most are also the students for whom the benefit is most difficult to exploit, since a short window demands tight coupling between the activity and the class intended to profit from it. A larger peak with a slower decay would mean the opposite, and would strengthen considerably the case for morning activity provision aimed at exactly those students.

Nothing in the current design convention allows the alternatives to be separated, because amplitude and persistence cannot be distinguished from a single measurement. Reporting an interaction between baseline standing and delay would be straightforward in a multi-timepoint design and is, so far as we are aware, unreported.

4.5 The applied question is a timetabling question

Universities do not need to know whether exercise improves executive function. They need to know how to arrange a timetable. Those are different questions, and only the first has been answered.

An institution deciding whether to schedule compulsory physical education in the morning rather than the late afternoon is asking whether a benefit conferred at nine o'clock is still present at eleven. A lecturer considering a five-minute activity break within a three-hour block is asking how much of the remaining block it will cover. A campus recreation service deciding opening hours, or a residential college deciding whether to place a gym beside the library, is asking the same question in a different form. Each is a question about persistence, and the literature's answer to each is currently an extrapolation from evidence that did not measure it.

The extrapolation is not neutral in its risks. If the effect is short-lived, then provision placed at a temporal distance from the target class may be delivering health benefits — which are substantial and independently justified (Bull et al., 2020) — while delivering no cognitive benefit at all; and evaluations of such provision may return null cognitive results that are misread as evidence against the acute effect rather than as evidence about scheduling. If the effect is longer-lived than assumed, then conservative arrangements that insist on immediate adjacency unnecessarily constrain what institutions can implement. Both errors are avoidable, and both are currently being risked.

The activity-break literature illustrates the problem in a setting where the temporal structure is unusually visible. A break is inserted into a teaching block precisely because the surrounding block is the target, so the relevant question is not whether the break improves cognition at its conclusion but how much of the subsequent session it covers. Evaluations nonetheless typically assess immediately after the break, which measures the peak rather than the coverage. A break producing a large improvement that lasts four minutes and one producing a smaller improvement that lasts thirty would be scored in the same direction, and the second would be scored lower — although in a two-hour seminar the second is plainly the more useful intervention.

4.6 Delay heterogeneity as a source of meta-analytic heterogeneity

Meta-analyses in this area routinely report substantial residual heterogeneity, which is then attributed to differences in population, protocol and outcome measure. Delay belongs on that list and is rarely on it.

The mechanism is straightforward. If effect magnitude varies with delay, and delay varies across studies, then delay contributes variance to the pooled estimate. Whether that contribution is large or small is an empirical question, and it is answerable: delay could be coded as a continuous moderator and entered into meta-regression, exactly as intensity and duration routinely are. The obstacle is not statistical but documentary. Meta-regression on delay requires that primary studies report delay precisely, and a substantial share do not — reporting that assessment occurred "after" the bout, or "following a short rest", or specifying an interval for the protocol as a whole without specifying the interval to the onset of each task in a battery.

That last detail deserves emphasis. When several tasks are administered in sequence, they occupy different positions on the decay curve. A battery lasting twenty minutes may place its

first and last tasks in materially different states. Reporting a single figure for the battery discards this information, and the ordering of tasks becomes an uncontrolled source of between-study variation.

There is a further consequence for cumulative science. Because delay is unmodelled, a replication attempt that assesses at a different delay from the original is not recognised as having altered a material parameter. It will be reported as a replication, and its result — whatever that result is — will be read as bearing on the original finding. Non-replication in this literature is therefore harder to interpret than it appears, since a failure to reproduce an effect may reflect a different sampling position on the curve rather than a fragile phenomenon.

4.7 Acute persistence and chronic adaptation are distinct questions

A final conflation deserves isolating: the relation between the acute effect and the effects of habitual activity or fitness. The two are frequently discussed together, and provision is routinely justified by evidence drawn from both, but they are different phenomena with different temporal structures.

The acute effect is a perturbation followed by a return towards baseline. Chronic adaptation is a shift in the baseline itself, accumulated over weeks or months and not expected to decay between sessions. A semester-long physical education course may in principle produce both: a transient elevation on each day it meets, and a gradual raising of the level to which that transient returns.

These components are separately identifiable in principle and almost never separated in practice. A semester course evaluated by a post-test administered shortly after the final session measures their sum. One evaluated some days later measures adaptation alone. Because evaluation timing is rarely reported with the precision that would allow the two designs to be told apart, apparently conflicting findings about course-length interventions may partly reflect whether the final assessment did or did not capture a residual acute effect.

The practical implication runs in both directions. Provision whose benefit is wholly acute must be sustained indefinitely to keep delivering it, and its effect will be absent in any week the activity does not occur — including, notably, the examination period, when timetabled physical education typically stops. Provision whose benefit is adaptive would be expected to persist across such gaps. These are materially different propositions for an institution deciding whether to protect timetable space, and current evaluations do not distinguish them. Reporting the interval between the final session and the post-test — a single number — would go a considerable way towards making the distinction recoverable.

5. Conclusion and recommendations

The acute effect of exercise on executive function is well established in its existence and almost entirely unspecified in its duration. This is a consequence of a design convention — one post-exercise measurement, at a variable and often unreported delay — that was reasonable when

the priority was to establish that an effect existed, and that has outlived its usefulness now that the priority is to use it.

The remedy has two parts, and neither is expensive.

5.1 A reporting minimum

Table 2 sets out ten items that would render future studies interpretable with respect to timing and, crucially, poolable. The central item is the first: the elapsed time from cessation of exercise to the onset of each cognitive task, reported in minutes rather than characterised in words. Most of the remaining items are already collected by most studies and simply not reported.

Table 2: A minimum reporting set for post-exercise cognitive assessment

No.	Item	Why it matters
1	Elapsed minutes from exercise cessation to onset of each cognitive task	The single quantity that locates the study on the decay curve
2	Order of tasks within the battery and duration of each	Later tasks occupy later positions on the curve
3	Whether the delay was fixed by protocol or varied by participant	Determines whether delay can be entered as a moderator
4	Activity performed during the interval between exercise and testing	Continued light activity may sustain arousal
5	Exercise intensity, verified by an objective index rather than prescribed only	Permits separation of intensity from delay
6	Exercise duration and modality	Standard, but frequently reported without the timing items above
7	Timing of any physiological or haemodynamic sampling relative to the same clock	Enables mechanism–effect time-course comparison
8	Time of day of the session	Diurnal variation in both arousal and executive function
9	Time since the participant's last meal and habitual sleep on the preceding night	Both modulate the effect and both vary with timetable
10	Baseline executive function of the sample, reported distributionally	Permits the amplitude-versus-persistence interaction to be tested

Source: Developed for this research.

Adopting these items imposes essentially no additional data collection burden. It would, within a few years, make possible the meta-regression on delay that cannot presently be conducted, and would allow the existing literature to be extended rather than merely repeated.

5.2 Design recommendations

Three design changes would move the field from point estimates towards functions.

First, multiple post-exercise assessments. Even two timepoints — one immediate and one delayed by a substantial interval — permit an estimate of direction and rough rate of change, and transform a scalar into the beginnings of a curve. Where repeated administration of the same task raises practice-effect concerns, parallel forms or counterbalanced task order across participants address the problem by established means.

Second, treatment of delay as a factor rather than a nuisance. Randomising participants to assessment delay, rather than fixing it, converts an uncontrolled source of variance into an estimable parameter and requires no additional participants beyond those a conventional design would recruit for the same power on the main contrast.

Third, joint measurement of a physiological or haemodynamic marker and cognition at each timepoint. Because the candidate mechanisms differ in time constant, the correspondence between the decay of a marker and the decay of the cognitive effect is far more informative about mediation than their correlation at one moment. This is where the theoretical return on multi-timepoint designs is greatest.

A note on statistical power is warranted, since the recommendation may appear costly. Adding timepoints within participants does not divide the sample; it adds observations to each participant, and where the within-participant correlation is appreciable it increases precision on the average effect while additionally yielding the slope. The design change is therefore closer to free than it looks, and its principal cost is session length rather than sample size.

5.3 Why higher education is the right setting to answer it

Universities are unusually well placed to conduct the studies this review calls for, and that is not incidental to the argument.

The population is accessible, cognitively assessable without the burden that repeated testing imposes on younger participants, and already timetabled — which means that assessment delays of thirty, sixty or ninety minutes can be scheduled rather than improvised. Compulsory physical education courses supply naturally occurring exercise bouts of known intensity and duration at known clock times, with a subsequent academic timetable that provides the very outcome window the applied question concerns. Brief computerised executive function tasks with established psychometric properties can be administered on portable hardware in a few minutes, making three or four assessments within a morning feasible. Ambulatory monitoring allows heart rate to be recorded continuously across the post-exercise interval, so that the decay of a physiological marker and the decay of a cognitive effect can be placed on the same clock.

None of this is exotic, and none of it requires resources beyond those a physical education department already has. A study of conventional size, using a battery it would have administered anyway, could estimate a rough decay curve by administering that battery at two

or three delays rather than one and randomising order across participants. The obstacle is convention rather than resource — and the institution that removes the convention answers its own scheduling question in the process.

5.4 Limitations and closing remark

This review is narrative and purposive, and its central empirical claim — that delay contributes materially to heterogeneity — is argued rather than demonstrated. Demonstrating it requires the meta-regression that current reporting does not support, which is the argument's point but also its principal weakness: the case for the reform rests partly on evidence that the reform would generate. We have also treated the mechanisms' time constants coarsely, and the balance of evidence across executive function components is uneven, with inhibition far better represented than updating or flexibility.

The broader observation is that a field can establish an effect thoroughly and still be unable to answer the question its applications depend on. The acute exercise literature has demonstrated that a bout of activity improves cognitive control in young adults. Whether that improvement is still there when the next lecture begins is a question of comparable practical importance to the institutions that timetable both, of greater theoretical discriminating power, and of markedly lower cost to answer than the question already settled.

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