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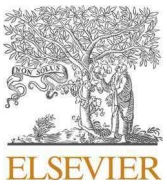

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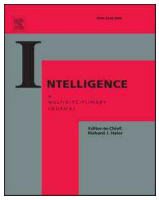

Signs of a Flynn effect in rodents? Secular differentiation of the manifold of general cognitive ability in laboratory mice (*Mus musculus*) and Norwegian rats (*Rattus norvegicus*) over a century—Results from two cross-temporal meta-analyses

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ABSTRACT

Substantial improvements in factors such as microbiological quality have been noted in laboratory rodent (mouse [*Mus musculus*] and rat [*Rattus norvegicus*]) populations over the last 140 years, since domestication of laboratory strains started. These environmental improvements may have caused Flynn effect-like cognitive changes to occur in these populations, perhaps if these improvements enhanced cognitive plasticity and, consequently, learning potential. While lack of relevant data precludes cross-temporal comparison of cognitive performance means of laboratory rodent populations, it is possible to estimate changes in the proportion of cognitive performance variance attributable to general cognitive ability (GCA) over time. This "differentiation effect" has been found to occur along with the Flynn effect in human populations, suggesting that environmental factors, possibly mediated by their effects on life history speed, may weaken the manifold of GCA across time, allowing for greater cultivation of specialized abilities. Meta-analysis of the literature on mouse and rat cognition yielded 25 mouse studies from which 28 GCA effect sizes could be estimated, and 10 rat studies from which 11 effect sizes could be estimated. Cross-temporal meta-analysis yielded evidence of significant "differentiation effects" spanning approximately a century in both mice and rats, which were independent of age, sex, factor estimation technique, and task number in the case of the mice, and both factor estimation technique and task number in the case of the rats. These trends were also independent of the random effect of strain in both cases. While this is suggestive of the presence of the Flynn effect in captive populations of non-human animals, there are still factors that might be confounding these results. This meta-analysis should be followed up with experimental investigation.

1. Introduction

The Flynn effect (named by Herrnstein & Murray, 1994 in honor of James Flynn, 1934–2020) is the tendency for performance on conventional IQ tests to improve over time across many different human populations. This improvement translates into an aggregate score increase of approximately three IQ points per decade, which seems to have occurred for roughly a century in at least some areas (Pietschnig & Voracek, 2015; Trahan, Stuebing, Fletcher, & Hiscock, 2014). Although other researchers had noted this tendency as far back as the 1930s (e.g., Runquist, 1936), James Flynn was the first to demonstrate compellingly the strong pervasiveness of this effect over relatively recent historical time and across populations (Flynn, 1984, 1987).

Thus far, research on the Flynn effect, and related phenomena, has focused exclusively on humans, and yet there is also a mass of data on cognitive performance in non-human animals. General cognitive ability (GCA), or GCA-like latent variables, have been found in the intercorrelations among a wide variety of individual differences measures used to assess cognition in numerous mammalian and avian species (for reviews, see Chabris, 2007; Burkart, Schubiger, & van Schaik, 2017; Poirier, Kozlovsky, Morand-Ferron, & Careau, 2020). GCA-like factors have even been found in Trinidadian guppies (*Poecilia reticulata*; Prentice, Mnatzaganian, Houslay, Thornton, & Wilson, 2022). These findings suggest that GCA might be very common among animals, with some

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hints that it may exist even in certain insect taxa (Chabris, 2007).

2. GCA in laboratory rodents

Especially well studied in this regard are laboratory rodents. There have been more studies into the structure of cognition in laboratory mice (*Mus musculus*) than any other (non-human) species (Burkart et al., 2017; Poirier et al., 2020). The earliest mouse study, from which GCA could be extracted, comes from the 1920s (Bagg, 1920),[1] with a great many more studies having been published between 1998 and the current day. Norwegian rats (*Rattus norvegicus*) are also reasonably well studied, with the earliest experiments designed explicitly to detect the presence of GCA having been conducted in the early 1930s (e.g., Commins, McNemar, & Stone, 1932; Thorndike, 1935). During this "first wave" of research into GCA in laboratory rodents (which lasted roughly from the second decade of the last century to the late 1930s), relatively poorly characterized samples were employed (these were often inbred, or mutant strains such as albinos). Moreover, it has been suggested that the tendency among early researchers to focus on prospectively narrower domains of cognition, such as spatial reasoning as measured by performance on different maze-running tasks, may have had the effect of limiting the degree to which their findings of GCA-like factors generalize relative to modern studies of GCA in laboratory rodents (Locurto, 1997).

A sort of "dark age of individual differences research" followed the "first wave" of GCA research in non-human animals, the former lasting from roughly the early 1940s to the late 1980s. A pronounced shift in research focus toward approaches such as behaviorism, with its emphasis on conditioning and learning, characterized this period. During this time, researchers had a tendency to argue that manifestations of animal ability were primarily dependent upon the use of specific apparatuses, and that there was no reason to anticipate correlations among distinct performance domains (e.g., Tryon, 1940; Warren, 1977; cf. Livesey, 1970; Rajalakshmi & Jeeves, 1968).

Following on from the "dark age" is what could be termed the "second wave" of research into GCA in laboratory rodents, which started in roughly the late 1980s and is ongoing today. This period has been characterized by efforts to broaden ability batteries so that they tap wider arrays of performance domains, which have been coupled with efforts to determine the psychometric soundness of such measures (Crinella & Yu, 1995). Paradigmatic of this approach is the study of Galsworthy, Paya-Cano, Monleón, and Plomin (2002), which detected GCA in mice using six tasks spanning several different domains. These included curiosity (as indexed by spontaneous alternation of direction in a T-maze), route learning (evaluated using the Hebb-Williams maze), spatial navigation (assessed using the Morris water maze), detour problem solving (evaluated using a burrowing task), contextual memory, and plug puzzle performance. In another example, Anderson (1993) acquired evidence of GCA in rats using four broad tasks. These tapped the domains of attention to novelty and accuracy of reasoning (evaluated using an eight-arm radial maze), as well as speed and response flexibility (evaluated using the detour problem).

As Crinella and Yu (1995) note, tasks administered to laboratory rodents with varying degrees of complexity fundamentally overlap with the hierarchy of GCA-loaded tasks in human psychometric assessments, in that more complex behavioral challenges and more GCA-loaded IQ test items both present their subjects with novel stimulus configurations necessitating an adaptive response in order to engage in effective problem solving.

[1] GCA was estimated to account for 61% of cognitive variance across eight tasks based on reanalysis of Bagg's (1920) published raw data in Galsworthy et al. (2005).

3. Laboratory rodents as a potential model for the study of the Flynn effect and related trends

Could laboratory rodents such as mice and rats be used for experimental investigations into the Flynn effect, or Flynn effect-like phenomena? There are two ways in which this could potentially be accomplished. First, experimental work involving these animals could be used to directly test theories of the causes and consequences of the Flynn effect. Both mice and rats are exceptionally well characterized in terms of behavior, neurology, and molecular biology, and are used as model organisms for experimental research in many branches of biology and medicine (Ellenbroek & Youn, 2016). They exhibit a variety of desirable characteristics that may qualify them as ideal model organisms for experimentally studying the Flynn effect, and related trends. These include:

i) **Sensitivity to gene-by-environment interactions**. It has been found that both trait variance and broad-sense heritability in mouse cognitive performance (including at the level of GCA) can be manipulated through environmental enrichment, via the action of gene-by-environment interactions (Matzel, Bendrath, Herzfeld, Crawford, & Sauce, 2019; Poirier et al., 2020).[2] Gene-by-environment covariation, and resultant interactions (Johnson & Bouchard Jr., 2014), is likely central to the etiology of the Flynn effect in humans (e.g., De Kort et al., 2014; Dickens & Flynn, 2001). By tightly homogenizing additive genetic variance through the use of highly inbred strains, or twins, and via the use of environmental manipulations designed to actively cognitively engage experimental subjects, it may be possible to maximize the degree to which trait variance is sensitive to gene-by-environment interactions in such a way that could be used to experimentally magnify the impacts of Flynn effect-salient environmental factors on cognitive performance across generations.

ii) **Fine control over environmental and genetic factors + short generation times**. Environmental and experimental conditions can be closely controlled in studies using mice and rats, which, along with short age-to-sexual-maturity times for both species (approximately four to seven weeks for mice and six to seven for rats), potentially allows for the multi-generational effect of a variety of prospective environmental causes of the Flynn effect to be experimentally investigated within a reasonable timeframe and in the absence of genetic confounds. These prospective environmental causes could be investigated via experimental manipulation of, for instance, nutrition quality, pathogen loads, and cognitive stimulation. The predictions of meta-causal models of the Flynn effect could also be tested, such as the life history model, which would involve the manipulation of more general factors such as environmental harshness and predictability (the life history model is discussed in greater detail in later sections). Multi-generational models would, further, enable evaluation of the degree to which given interventions may be associated with an especially striking aspect of the Flynn effect, specifically that it is cumulative and persistent, and therefore does not exhibit the

[2] It should be noted that Sauce et al. (2018) were unable to find any indications of gene-by-environment interactions on cognitive ability in experimental research that involved exposing mice to environments that promote cognitive development, despite noting a 0.44 standard deviation, or 6.6 "IQ point" increase, in the trait (relative to controls).The environmental manipulations employed in this study may have been too subtle to yield measurable gene-by-environment interactions. In a subsequent analysis of the same dataset of mouse twins, Matzel et al. (2019) found evidence of substantial gene-by-environment interactions on measures of learning associated with exploration, in which the animals were actively "challenged" in such a way that experimentally better "draws out" such interactions.

"fadeout effect" that usually follows targeted environmental interventions to boost cognitive ability in human populations (Protzko, 2015). Moreover, experimental work could also explore potential environmental causes of anti-Flynn effects (e.g., Bratsberg & Rogeberg, 2018).

iii) **Molecular toolkit.** The availability of a robust molecular toolkit for both mice and rats (in particular for the former, Ellenbroek & Youn, 2016) allows the epigenetic influences of the aforementioned environmental effects to potentially be directly mapped onto alterations in patterns of gene expression.

iv) **Neural mapping of behavior**. The ability to use neural lesioning and other techniques to map behavior onto neuroanatomy in laboratory rodents might allow for theories of the neural localization of the Flynn effect to be directly tested, such as those positing a role for the secular expansion of the right hippocampal formation in humans (Baxendale & Smith, 2012).[3]

v) **Ratio-scale ability measures**. Cognitive data on laboratory rodents are gathered with ratio-scale measures, which side-step the problems for time-trend analysis that interval-scale measures present, since the latter lack a "true" zero performance baseline for the estimation of ability trends over time (as noted by Jensen, 2011).

vi) **Tests of models positing mixed time trends**. Tests of models positing co-occurrent, and opposingly signed, trends in different variance components of human IQ (such as those postulating genetically driven decreases in GCA, coupled with environmentally driven increases in more specialized abilities; Egeland, 2022), or those positing a role for reductions in autozygosity associated with heterosis in the Flynn effect (Mingroni, 2007), could also be tested with experimental designs in which directional selection or degree of inbreeding are used to induce changes in GCA and related phenotypes and traits (e.g., processing speed or white matter integrity), against a backdrop of environmental change(s) designed to alter levels of less heritable, narrower abilities in opposing ways.

4. Examination of time trend data using existing cognitive data on mice and rats

A second approach to investigating the Flynn effect in laboratory rodent populations involves comparative analysis of existing studies. As already mentioned, laboratory rodents, and mice in particular, are reasonably well studied in terms of the identification of GCA, or GCA-like latent variables, with individual differences studies dating back to the 1920s in the case of mice, and the 1930s in the case of rats. Moreover, there are a series of well-characterized historical developments in the animal husbandry of these experimental populations that are suggestive of the presence of the sort of environmental improvements believed to have elicited the Flynn effect in human populations. One such example is discussed by Buchheister and Bleich (2021), who have identified four key phases in the historical husbandry of laboratory rodents which encompass improvements in health monitoring and microbiological quality. Phase 1 corresponds to domestication (1880–1950), phase 2 to gnotobiotic derivation (1960–1985), phase 3 to virus eradication (1980–1996), and phase 4 to isolated husbandry (current period). The transitions between these phases can be described in relation to questions concerning improvements in husbandry and experimental control. The initial challenge is captured by the question, "how do we keep them alive?"; this was followed by "how do we keep them healthy?", then by "how do we ensure their quality?", and finally "how do we ensure (strain) validity?" Improvements in the "microbiological quality" of humans (e.g., via the historical control and eradication of infectious and parasitic diseases) is thought to have played a major role in catalyzing the Flynn effect, by virtue of having allowed for the reallocation of bioenergetic resources, which would otherwise have been used to boost immune functioning, into the cultivation of cognitive abilities (Eppig, Fincher, & Thornhill, 2010). The historical process described by Buchheister and Bleich (2021) may have had similarly beneficial effects on the cognitive ability of populations of laboratory rodents.

Other sorts of environmental improvements might have also contributed to the Flynn effect in laboratory rodent populations. These might include increased environmental enrichment, dietary improvements, and other environmental changes occasioned in part by the passage of animal welfare legislation in a variety of countries, ensuring that laboratory animals are treated in a more humane way when used for behavioral research (e.g., Guidelines for the Use of Animals, 2018). Substantial variation in a variety of phenotypes, including brain size, between laboratories and over time, has been noted in various strains of laboratory mice (Wahlsten, Bachmanov, Finn, & Crabbe, 2006). These findings might suggest the occurrence of environmentally influenced time trends (in cases where this variation occurs over time).

A major shortcoming in the existing cognitive data on mice and rats is the substantial heterogeneity among tests used to measure cognition. This coupled with the fact that many studies do not in fact report performance averages, only correlation matrices or factor loadings, prevents efforts to estimate mean performance change over time. The most direct approach to establishing a Flynn effect (via comparison of performance means over time) therefore cannot be taken with these data.

5. The life history model of the Flynn effect and ability differentiation

Among human populations, it has been hypothesized that different sorts of environmental improvements might have distinct effects on secular ability gains, with these improvements having played different roles at different times (Williams, 2013). For example, among relatively malnourished populations, improved nutrition may have the biggest role in generating the Flynn effect, whereas among adequately nourished populations experiencing expanded access to education, the latter factor might play the major role. An alternative hypothesis is that there are common pathways through which a variety of environmental improvements can act. The (currently) best supported model of the Flynn effect aiming to theoretically integrate this variety of causes (hence it might be called a "meta-causal" model) holds that the effect is a developmental product of life history (LH) tradeoffs favoring enhanced cognitive plasticity and specialization as an adaptation to increased population densities at the expense of forms of cognition favoring greater preparedness for the sorts of environmental stresses that reduce population densities (as reflected in greater cognitive generalism) (Pietschnig & Voracek, 2015; Woodley, 2012).[4] These tradeoffs are likely to have been occasioned by the historical slowing of LH speed noted in human populations, which has resulted from modernization having reduced both the absolute levels of, and variance in sources of, extrinsic (uncontrollable) morbidity and mortality, increasing environmental mildness and predictability (Baumard, 2019). More broadly, slower LH is associated with greater somatic effort (i.e., bioenergetic investments in the organism's own survival, which may be reflected in enhanced body growth, such as greater stature, and mental and physical health) and parental/nepotistic effort (i.e., bioenergetic investments that prospectively increase the fitness of those with relatively high

[3] Kempermann, Kuhn, and Gage (1997) have noted that hippocampal neuron count is increased among mice exposed to enriched environments.

[4] A similar meta-causal model of the Flynn effect termed the *cognitive genome optimization hypothesis*, has been proposed by Greiffenstein (2011), who suggests that "[p]er life-history theory, favorable secular trends may change the phenotypic expression of the genotype which controls the neurophysiology of problem solving" (p. 353).

genetic relatedness to the organism). It is also associated with reduced mating effort (i.e., bioenergetic investments in the acquisition and retention of short-term sexual partners) (Figueredo, Vásquez, Brumbach, & Schneider, 2004). More weakly correlated cognitive abilities have been found in individuals exhibiting slower LH (Woodley, Figueredo, Ross, & Brown, 2013). This suggests that an increased capacity for cognitive specialization in such individuals might be another important consequence of increased somatic and/or parental/nepotistic effort (Woodley et al., 2013).

The LH model aims to account for the following Flynn effect-associated observations:

i) **Demographic transition:** The Flynn effect coincides with the onset of the demographic transition, during which various trends suggestive of increased somatic effort began, such as increasing stature, longevity, and even brain mass in some populations (Mingroni, 2007; Pietschnig & Voracek, 2015; Woodley of Menie, Peñaherrera, Fernandes, Becker, & Flynn, 2016),
ii) **Lower time preferences:** Time trends paralleling the Flynn effect over several decades suggest a lowering of time preferences (Protzko, 2020), which is consistent with increasing self-control. Higher self-control is a key feature of behavioral manifestations of slower LH (Figueredo et al., 2004).
iii) **Differentiation effects:** Secular gains in IQ are often accompanied by ability differentiation, i.e. a reduction in the strength of the GCA factor (specifically as measured by a reduction in the proportion of variance in cognitive performance accounted for by the first dimension, or mean intercorrelation strength, among subtests in more recently born cohorts relative to older ones). These effects have been noted in a number of studies of the Flynn effect (for a review of this literature, see Woodley & Madison, 2013).[5] As previously noted, slower LH seems to be associated with weaker associations among cognitive abilities (Woodley et al., 2013). Woodley and Madison (2013) found evidence of a direct relationship between ability differentiation and the Flynn effect using three administrative waves of the Estonian National Intelligence Test—a significant association was found between the decrease in the GCA loading of subtests across waves and the magnitude of performance gains on subtests. This, it was argued, is consistent with the idea that the Flynn effect involves differential, or specialized, rather than uniform gains across abilities, with more GCA-loaded (and prospectively also more heritable) ability measures being notably more weakly associated with the effect (for more discussion, see te Nijenhuis & van der Flier, 2013).

Unlike changes in performance over time, the presence of a differentiation effect over time *can* be tested in laboratory rodents given the available data on GCA proportion of variance. The presence of this effect in populations of laboratory rodents furthermore may be suggestive of broader LH slowing in response to the various environmental improvements noted before, and are at least suggestive of the presence of a Flynn effect in these populations, as they might result from increasing cognitive plasticity and specialization. At the level of test performance, this might be expected to manifest as a progressively greater affinity for learning on certain tasks as evidenced by reductions in the latency of learning periods (e.g., fewer trials would be needed for successful task completion among more recent cohorts). Fig. 1 highlights the presumed sequence through which various forms of environmental improvement might translate into performance gains among laboratory rodents.

In the current study, a cross-temporal meta-analysis is conducted in order to determine whether changes in the strength of GCA (specifically differentiation effects, which entail a reduction in GCA-associated variance) have occurred in laboratory populations of mice and rats. Data on a number of confounding moderator variables will also be collected in order to ascertain the robustness of these effects, if present.

6. Methods

6.1. Meta-analytic search strategy

As (non-human) animal studies from which GCA can be estimated are reasonably uncommon, somewhat "generous" inclusion rules were applied in order to maximize the number of studies available for analysis. For a study to be included, it needed to meet the following five criteria:

i) The subjects must either be laboratory mice (*Mus musculus*) or Norwegian rats (*Rattus norvegicus*).
ii) The sample size must exceed five individuals.
iii) The number of ability measures must exceed two.
iv) The subjects must be healthy.
v) Sufficient data to allow for factor estimation must be available (this can take the form of raw data, correlation matrices, and/or factor estimation conducted as part of the original study).

The basis of the literature search was the meta-analysis of Poirier et al. (2020), which examined the prevalence of GCA across non-human animals. These researchers searched Scopus and Web of Science using the Boolean string "general cogniti*" OR "general intel*". The results of their search were supplemented by consulting two recently published non-systematic reviews of the (recent) literature on GCA in animals (Burkart et al., 2017; Flaim & Blaisdell, 2020). None of these literature reviews considered the older literature on GCA in animals, specifically that from the early decades of the 20th century. These early data were however comprehensively reviewed quantitatively in Chabris (2007, with factor estimation), and qualitatively in Galsworthy, Arden, and Chabris (2014, without factor estimation). Finally, Google Scholar was searched in order to identify more recently published, or lower visibility (not listed on Scopus or Web of Science), studies that might contain relevant data. This was conducted using the following four strings: "general cognitive" AND mice, "general cognitive" AND rats, "general intelligence" AND mice, and "general intelligence" AND rats.

6.2. Sample of studies

The current study followed the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) recommended guidelines (Moher, Liberati, Tetzlaff, & Altman, 2009). The steps used in reducing the initial study pool (75) to the final pool (35) are reported as a flow diagram in Fig. 2.

All but two of the studies identified adhered to the inclusion rules. For the most part, sample sizes were substantially greater than five (the smallest sample size was eight rats in the case of Livesey, 1970). There was substantial redundancy among literature reviews due to overlapping inclusion of studies, however, with most "exclusions" being duplicates. The study of Kolata, Light, and Matzel (2008) was also excluded as a (partial) duplicate on the basis of textual review. This

[5] Lynn and Cooper (1993) were perhaps the first to predict the existence of this temporal differentiation effect on the basis that gains in IQ over time should be associated with Spearman's Law of Diminishing Returns (SLODR), that is the tendency for GCA to account for a smaller portion of cognitive variance (as evidenced by weaker subtest intercorrelations) among those with higher vs. lower IQs (for a recent meta-analysis of this effect, see Blum & Holling, 2017). Both SLODR and the Flynn effect share certain psychometric properties in common—for example, both effects appear to be more pronounced on subtests onto which GCA loads more weakly (Jensen, 2003; te Nijenhuis & van der Flier, 2013). This hints at a role for factors common to the environments of both higher-IQ individuals and members of more modernized populations in the etiology of the Flynn effect, consistent with speculations first advanced by these researchers.

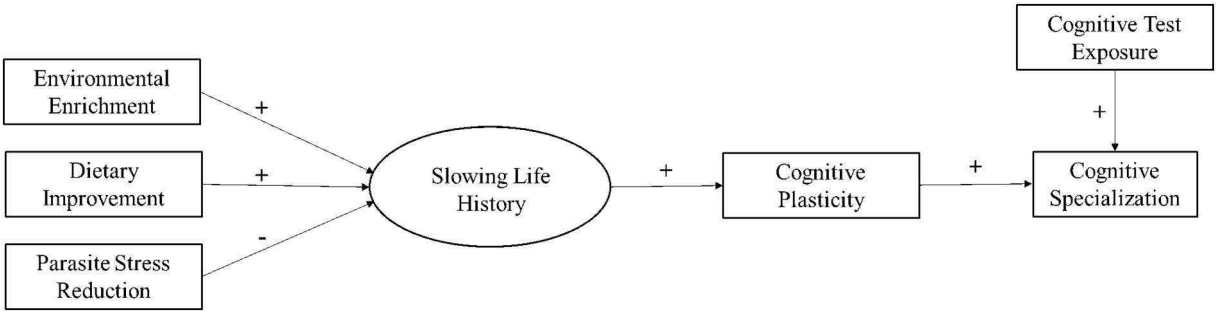

Fig. 1. Hypothetical sequence in which three sources of environmental improvement have effects mediated by slowing LH on cognitive plasticity, which yields enhanced cognitive specialization in response to exposure to certain cognitive tasks.

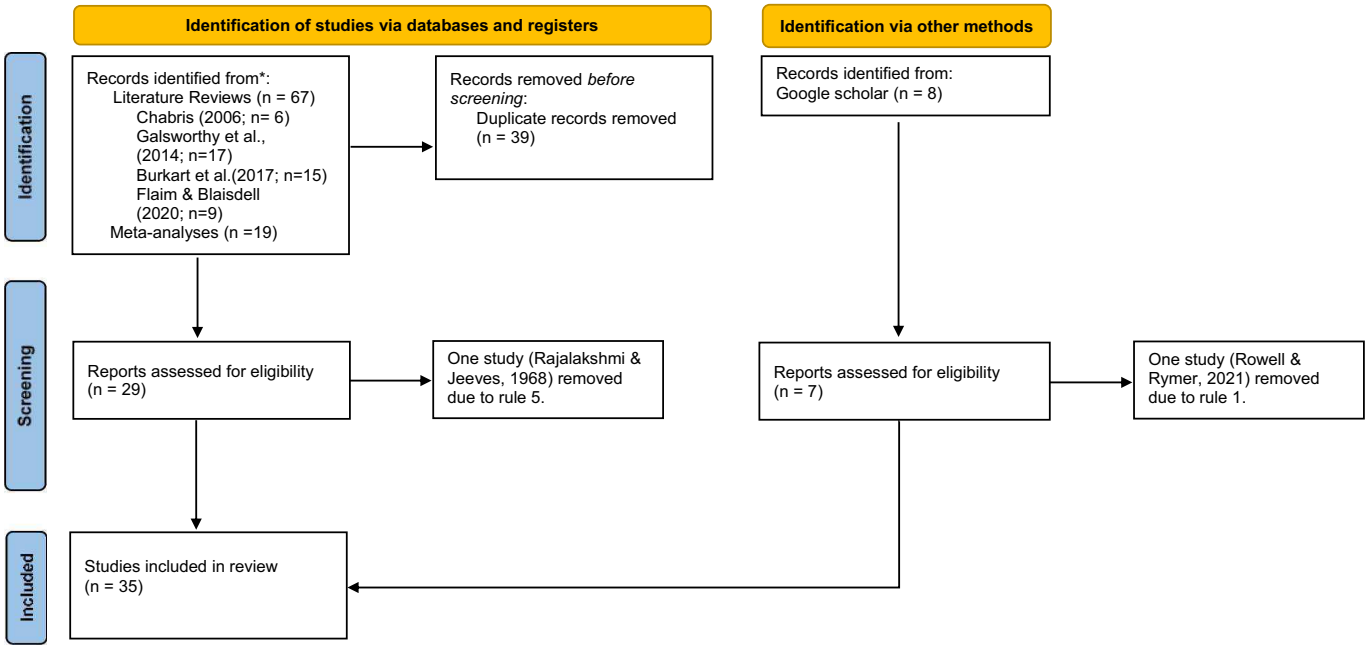

Fig. 2. PRISMA flow diagram illustrating the inclusion and exclusion of studies collected from various online databases and previously published literature reviews.

study combined data from four previous studies (Matzel et al., 2003 [$n = 56$]; Matzel et al., 2006 [$n = 43$]; Kolata et al., 2005 [$n = 21$]; Kolata, Light, Grossman, Hale, & Matzel, 2007 [$n = 27$]) with an additional 94 mice from unpublished studies, yielding a total sample size of 241. As the results were not broken out by sample, this study could not be used as the data were (for the most part) non-independent with respect to other studies that were identified in the meta-analytic search.

One rat study that was included among the studies reviewed in Galsworthy et al. (2014) on the basis that it contained information on three or more measures of cognitive ability was that of Rajalakshmi and Jeeves (1968). This study was however excluded from the current meta-analysis on the basis of the fifth inclusion rule, specifically it did not contain enough information on the relevant correlations so as to allow for factor extraction. Another study was identified on the basis of the Google Scholar search that identified GCA in a different species of rat (*Melomys cervinipes*; Rowell & Rymer, 2021). This study was excluded on the basis of the first inclusion rule. Some of the studies examined cognitive performance and covariance in rodents that had been experimentally lesioned (Anderson, 1993, 1995; Crinella & Yu, 1995; Thompson, Crinella, & Yu, 1990). In these cases, only data on the unlesioned healthy controls were retained for analysis (based on the fourth inclusion rule).

In total 25 suitable studies involving mice were identified, yielding 28 effect sizes; 10 suitable studies involving rats were identified, yielding 11 effect sizes.

In order to increase model realism, data collection years were used instead of publication years. Textual review of the collected studies yielded no information on data collection year, except in the case of Livesey (1970), where the data appear to have been collected in the early 1960s (Livesey's sample of eight "white rats" was first characterized in a study published in 1965 [Livesey, 1965]). A commonly used formula for approximating data collection years in cross-temporal meta-analysis is to subtract two years from the publication year of published papers and books, and one year from the publication year of "grey literature" (such as white papers and preprints), this being the typical lag between data collection and publication in each case (Pietschnig & Gittler, 2015; Twenge et al., 2010). On this basis two years were subtracted from the publication years of all studies except for Livesey (1970), where two years were subtracted from 1965—the year in which this sample was first characterized in the literature.

Data on several prospective moderators were also collected in order to control for factors that might confound possible temporal effects on the proportion of variance accounted for by GCA. These included subject age (in days), sex (coded as male, female, or mixed), strain type (if not specified this was given the label of "unknown"), the number of tasks comprising the GCA factor, and the factor estimation method (Principal

Axis Factor Analysis, PAF; or Principal Component Analysis, PCA). Subject age may affect the proportion of variance accounted for by GCA, as there are some indications from human studies of age-based differentiation and even de-differentiation effects (McArdle, Ferrer-Caja, Hamagami, & Woodcock, 2002, cf. Juan-Espinosa et al., 2002). It is possible that sex might also influence GCA proportion of variance, if there are sex differences in the mean inter-correlation strengths among cognitive abilities due to differential susceptibility to factors that might induce differentiation. Some evidence consistent with this in human populations has been found by Escorial, García, Juan-Espinoza, Rebollo, and Colom (2008), who noted that differentiation effects (by ability level) were absent among their female subjects, but were present among their male subjects. Task number may also to some extent proxy the nomological breadth of a given battery, which may also influence the estimation of latent variables (Jensen, 1998). As Principal Component Analysis and Principal Axis Factor Analysis deal with error structure in different ways, it is conceivable that the use of these different factor estimation techniques may moderate GCA variance proportions, especially when sample size, or indicator number, is low (Gorsuch, 2014). Finally, Crinella and Yu (1995) have suggested that strain might influence the degree to which GCA is present across studies of rodents. This variable was modeled as a random effect, meaning that it is possible to generalize the results of the moderator analysis with respect to the 'universe' of strains, including unmodelled ones.

All data employed in the cross-temporal meta-analyses are summarized in Table 1.

Two separate cross-temporal meta-analyses were conducted using mouse and rat data respectively. Far more effect sizes were available for mice than for rats (28 vs. 11), which is consistent with the observation that generally more research effort has been expended on the former (Ellenbroek & Youn, 2016). More moderators were available for the former relative to the latter also (five vs. three).

Interestingly, plotting out study number by year yields scientometric indications of the two research "waves" plus "dark age" model posited in the introduction. This is graphed in Fig. 3.

6.3. Statistical analyses

GCA variance was operationalized as the proportion of variance explained by the first unrotated component or factor, this corresponding to the dimension accounting for the most variance among all linear combinations of independent variables. When estimated using correlation matrices, PAF was used as the basis for factor estimation.

These proportions of variance were recomputed as Pearson's *r* coefficients later to be transformed into *z*-values using Fisher's *r*-to-*z* transformation. This last step allowed study effect size to be weighted by sample size. These transformations were implemented using the meta-analytic platform *OpenMee* (Wallace et al., 2017). A random effects meta-analysis, with maximum likelihood estimation and strain modeled as a random effect, was conducted to determine the moderating influence of sex (contrasting studies involving both males and females to studies limited to males), average age of individuals, type of latent variable modeling method used (contrasting PAF with PCA), number of cognitive tasks presented to the individuals, and data collection year, on the Fisher *z*-transformed GCA coefficients for studies conducted with mice. A similar model was computed for studies conducted with rats to determine the independent influences of number of cognitive tasks employed, factor estimation technique, and data collection year, on the Fisher *z*-transformed GCA coefficients. Sex and average age of individuals were not included in the meta-analysis involving rats because these data were not available for all studies. Meta-regression plots with 95% confidence intervals were produced to visualize the relationship between data collection year and the Fisher *z*-transformed GCA coefficients.

Meta-analytic examinations are often represented by two main figure types: forest plots and funnel plots. A forest plot is a graphical representation of the various effect sizes and their corresponding confidence intervals (Borenstein, Hedges, Higgins, & Rothstein, 2021). A funnel plot is a graphical representation of the effects under examination organized alongside a scale, generally following the *x*-axis, relative to a metric of their accuracy or precision (e.g., their standard errors) represented on the *y*-axis (Schwarzer, Carpenter, & Rücker, 2015). Funnel plots feature the studies' effects scattered in proximity to an average effect. If the database under consideration is characterized by a limited degree of between-study heterogeneity, smaller studies tend to be more distant from the average (display larger standard errors) (Schwarzer et al., 2015). Hence, a funnel plot follows a triangular shape with large and precise studies at the top of the figure and small and imprecise studies at the bottom. Asymmetrical funnel plots also indicate whether some studies might be missing from the statistical examination (Schwarzer et al., 2015). The current paper features both forest and funnel plots depicting the degree of effect size dispersion, the estimated mean effect size, and the extent of missing cases throughout these analyses.

In addition to generating these graphical representations the "trim and fill" procedure (Duval & Tweedie, 2000) was used to investigate the extent of potential publication bias due to the exclusion of unidentified studies altering the symmetrical distribution of effect sizes around the mid-line. Trim and fill operates as an iterative procedure that eliminates small and extreme values, usually from the right side of the funnel plot (Borenstein et al., 2021). Furthermore, each iteration re-estimates the effect sizes until the plot becomes symmetrical (Borenstein et al., 2021). *Trimming* reduces the effects' variance, providing adjusted estimates and unbiased effect sizes. *Filling* incorporates the original effect sizes into the model creating a mirror image (Borenstein et al., 2021). Similarly, Egger's regressions were carried out in order to quantify the degree of funnel plot asymmetry. These analyses were conducted with the package *metafor* (Viechtbauer, 2010) in *R* version 4.0.1.

7. Results

7.1. Cross-temporal meta-analysis of GCA variance in laboratory mice

The mouse cross-temporal meta-analysis, with strain as a random effect, yielded a pooled Fisher *z*-transformed coefficient of 0.657 (*95% CI* = 0.591, 0.723; $p < .0001$), equivalent to an R^2 of 0.322. This indicates that across studies, GCA accounts for 32.2% of the variance in cognitive performance. Fig. 4 presents a forest plot of the *z*-transformed GCA coefficients per study. As indicated by Table 2, the trim-and-fill model did not detect any missing studies. A funnel plot provided additional evidence for this finding (Fig. 5). It is worth noting that the Egger's regression test did reach statistical significance suggesting the presence of a funnel plot asymmetry, a result expected given the fact that three studies feature large effect sizes and large standard errors. The multivariate cross-temporal meta-analysis revealed that the model's residual heterogeneity was not statistically significant. Alternatively, the moderator test detected significant levels of heterogeneity. Concerning the various moderators, the analysis found no effects on GCA variance stemming from age, sex (the combined male and female studies were used as the reference category), number of tasks, or latent modeling method used (studies employing PAF were used as the reference category). In contrast, (critically) year (data collection) was associated with negative and significant influences on GCA variance. These results suggest that the proportion of variance explained by GCA has decreased over around 100 years in mice, net of confounds (Table 3). Fig. 6 plots the proportion of variance associated with GCA as a function of data collection year. The study of Bagg (1920) was somewhat of a visual temporal outlier (it seems there are no subsequent studies on mouse cognition yielding indications of GCA until 1998). As a robustness check, the meta-analysis was rerun excluding Bagg (1920). This yielded a pooled effect size of 0.629 (*95% CI* = 0.569, 0.689; $p < .0001$), equivalent to an R^2 of 0.310. Moreover, the meta-analytic moderation analysis

Table 1
Studies included in the current cross-temporal meta-analyses, along with effect sizes and relevant sample characteristics.

Study ID	Data collection year	Species	N	Strain	Mean Age (days)	Sex	Tasks	Statistics	R^2
Bagg (1920)	1918	*Mus musculus*	71	Hybrid Strain (HS)/Mixed	28	MF	8	PCA	0.61
Colas-Zelin et al. (2012)	2010	*Mus musculus*	48	CD1	68	M	5	PCA	0.29
Crawford et al. (2020)	2018	*Mus musculus*	74	CD1	35	M	6	PCA	0.38
Galsworthy et al. (2005)	2003	*Mus musculus*	167	C57BL/6 + BALB/c + RIII+AKR + DBA/2 + I + A/J + C3H	103.5	MF	6	PAF	0.18
Galsworthy et al. (2002)	2000	*Mus musculus*	40	C57BL/6 + BALB/c + RIII+AKR + DBA/2 + I + A/J + C3H	69	MF	6	PCA	0.31
Kolata et al. (2005)	2003	*Mus musculus*	21	CD1	90.3	M	5	PCA	0.34
Kolata et al. (2010)	2008	*Mus musculus*	60	CD1	100	M	5	PCA	0.41
Kolata et al. (2007)	2005	*Mus musculus*	27	CD1	90.3	M	5	PAF	0.37
Light et al. (2010)	2008	*Mus musculus*	29	CD1	55	M	5	PCA	0.30
Light, Kolata, Hale, Grossman, and Matzel (2008)	2006	*Mus musculus*	32	CD1	79	M	5	PCA	0.27
Light, Grossman, Kolata, Wass, and Matzel (2011)	2009	*Mus musculus*	24	CD1	50.5	M	5	PCA	0.41
Locurto, Benoit, Crowley, and Miele (2006)	2004	*Mus musculus*	20	HS/Mixed	127.5	M	5	PCA	0.28
Locurto et al. (2006)	2004	*Mus musculus*	35	HS/Mixed	127.5	M	5	PCA	0.34
Locurto, Fortin, and Sullivan (2003)	2001	*Mus musculus*	60	HS/Mixed	82.5	MF	6	PCA	0.19
Locurto and Scanlon (1998)	1996	*Mus musculus*	34	C57BL/6 + DBA/2Js	92.5	M	5	PAF	0.61
Locurto and Scanlon (1998)	1996	*Mus musculus*	41	CD1	84	MF	5	PAF	0.55
Matzel et al. (2006)	2004	*Mus musculus*	24	CD1	85	M	5	PCA	0.32
Matzel et al. (2020)	2018	*Mus musculus*	56	CD1	94.5	M	7	PAF	0.29
Matzel, Grossman, Light, Townsend, and Kolata (2008)	2006	*Mus musculus*	58	Balb/C	390	MF	5	PCA	0.31
Matzel et al. (2003)	2001	*Mus musculus*	56	CD1	83	M	5	PCA	0.38
Matzel, Kolata, Light, and Sauce (2017)	2015	*Mus musculus*	64	CD1	100	M	4	PAF	0.28
Matzel et al. (2011)	2009	*Mus musculus*	24	CD1	90.3	M	5	PCA	0.37
Sauce et al. (2018)	2016	*Mus musculus*	232	CD1	38.5	M	5	PAF	0.20
Sauce, Wass, Smith, Kwan, & Matzel, (2014)	2012	*Mus musculus*	26	CD1	90	M	4	PAF	0.44
Smith et al. (2013)	2011	*Mus musculus*	58	CD1	70	M	4	PCA	0.35
Wass et al. (2012)	2010	*Mus musculus*	41	CD1	59	M	5	PCA	0.29
Wass, Sauce, Pizzo, and Matzel (2018)	2016	*Mus musculus*	98	CD1	89	M	4	PCA	0.31
Wass et al. (2012)	2010	*Mus musculus*	25	CD1	59	M	5	PCA	0.32
Anderson (1993)	1991	*Rattus norvegicus*	22	Long-Evans	123	M	4	PAF	0.32
Anderson (1995)	1993	*Rattus norvegicus*	41	Long-Evans	67	M	3	PAF	0.26
Campbell (1935)	1933	*Rattus norvegicus*	28	Uknown	180	F	3	PAF	0.88
Commins et al. (1932)	1930	*Rattus norvegicus*	152	Uknown			4	PAF	0.92
Crinella and Yu (1995)	1993	*Rattus norvegicus*	24	Sprague-Dawley			7	PCA	0.28
Kassai, Ernyey, Kozma, Plangár, and Gyertyán (2022)	2020	*Rattus norvegicus*	36	Lister-Hooded		M	7	PCA	0.22
Kassai et al. (2022)	2020	*Rattus norvegicus*	36	Long-Evans		M	7	PCA	0.27
Livesey (1970)	1963	*Rattus norvegicus*	8	'White rats'		M	4	PAF	0.82
Thompson et al. (1990)	1988	*Rattus norvegicus*	75	Sprague-Dawley		M	4	PAF	0.43
Thorndike (1935)	1933	*Rattus norvegicus*	64	Albino			32	PAF	0.27
Tomlin and Stone (1934)	1932	*Rattus norvegicus*	132	Albino	100	MF	6	PAF	0.62

Note: R^2: The proportion of variance explained by either the first unrotated factor or the first principal component.

remained consistent with the previous results, with the reduced model also identifying a significant negative influence of year on the criterion variable ($b = -0.014$; *95% CI* $= -0.024, -0.003$; $p = .0116$). The negative time trend with respect to GCA variance in the reduced model is graphed in Fig. 7.

7.2. Cross-temporal meta-analysis of GCA variance in Norwegian rats

The second cross-temporal meta-analysis examined changes in GCA variance in rats over time. As with the previous analysis involving mice, strain was used as a random effect. A pooled Fisher *z*-transformed coefficient of 0.945 (*95% CI* $= 0.642, 1.249$; $p < .0001$) was found, equivalent to an R^2 of 0.545. This means that 54.5% of the variance in cognitive performance in rats can be attributed to the action of GCA. Fig. 8 provides a forest plot of the *z*-transformed GCA coefficients in each of the 10 studies. Neither the trim-and-fill nor Egger's regression test yielded any indications of funnel plot asymmetry, with the number of "missing" studies estimated to be zero (Table 4). Consequently, both the adjusted and unadjusted mean effect sizes are identical. The results are presented in Fig. 9. The multivariate cross-temporal meta-analysis

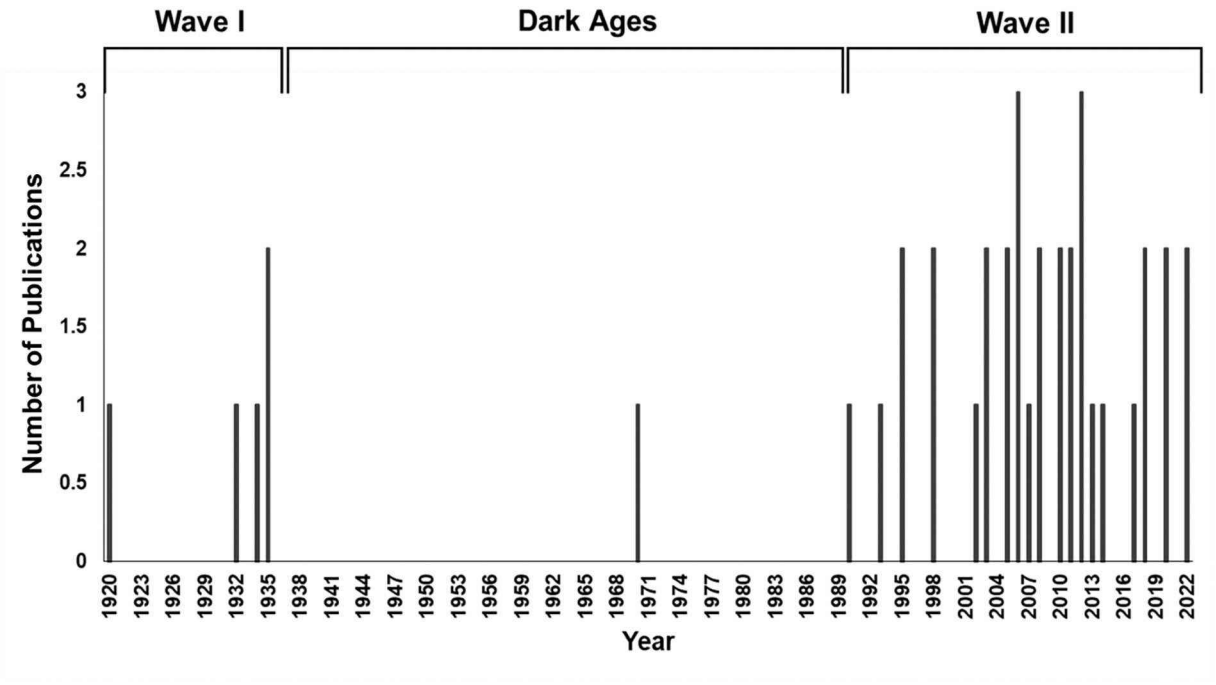

Fig. 3. Scientometric trend illustrating the number of publications (mice and rats combined) included in the cross-temporal meta-analyses by publication year.

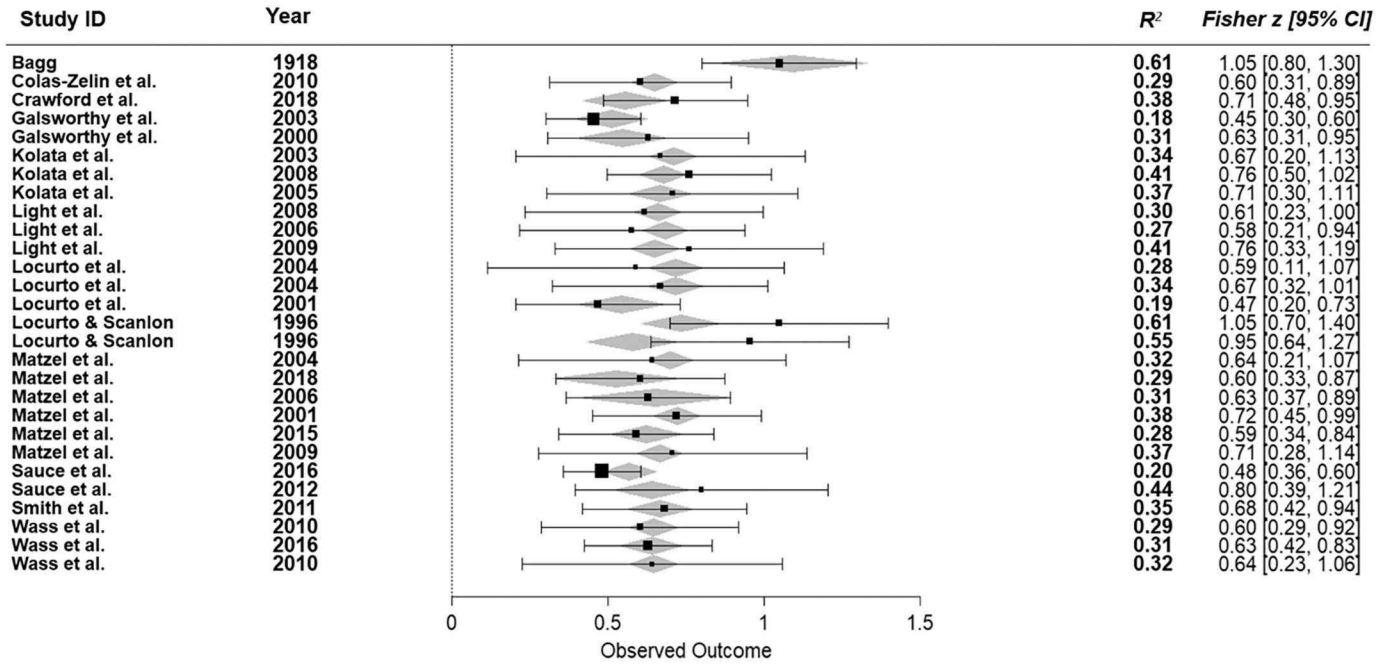

Fig. 4. Forest plot with *z*-transformed GCA coefficients across a sample of 28 GCA variance effect sizes in mice along with data collection years. The plot was generated using the *rma* function and the R package *metafor*.

Table 2
Results of the trim-and-fill test and the Egger's regression analysis of symmetry conducted with a sample of 28 GCA variance effect sizes in mice.

Trim-and-Fill				
Studies trimmed	*Point estimate*	*95% CI*	*p-value*	*Q*
Observed values = 27	0.657	0.591, 0.723	$p < .0001$	36.335
Adjusted values = 27	0.657	0.591, 0.723	$p < .0001$	36.335
Regression Test for Funnel Asymmetry				
Estimate	*95% CI*	*t-value*	*Df*	*p-value*
0.418	0.263, 0.573	2.99	26	0.0060

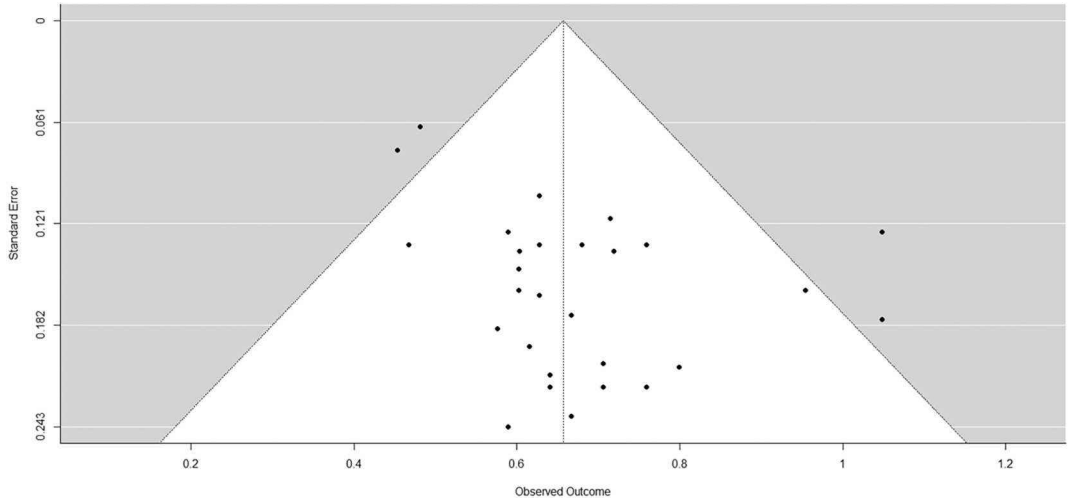

Fig. 5. Funnel asymmetry plot with trim-and-fill imputed "missing" studies (unfilled circles—in this case there were no missing studies) evaluating the absence of data points of *z*-transformed GCA variance coefficients across a sample of 28 effect sizes in mice. The graphic was generated with the R package *metafor*.

Table 3
Results of a multivariate cross-temporal meta-analysis examining the moderating effects of sex, age, latent variable modeling method, number of tasks, and year on the proportion of variance explained by GCA in mice across 28 effect sizes.

Multivariate Meta-Analysis Model (k = 28; method: ML)					
Test for Residual Heterogeneity:					
QE(df = 22) = 16.138, p-value = .8090					
Test of Moderators (coefficients 2:6):					
QM(df = 5) = 2.197, p-value = .0011					
Parameters (reference category)	*Estimate*	*95% CI*	*SE*	*z-value*	*p-value*
Intercept	15.651	(7.752, 23.550)	4.030	3.88	0.0001
Sex (Male and Female)	−0.154	(−0.326, 0.018)	0.088	−1.76	0.0787
Age	0.000	(−0.001, 0.001)	0.001	0.80	0.4225
Latent Variable Modeling Method (PAF)	−0.027	(−0.140, 0.087)	0.058	−0.46	0.6437
Number of Tasks	−0.024	(−0.107, 0.058)	0.042	−0.58	0.5644
Year	−0.007	(−0.011, −0.004)	0.002	−3.77	0.0002
Fit indicator	***logLik***	***Deviance***	***AIC***	***BIC***	***AICc***
Value	18.310	16.138	−22.620	−13.294	−17.020

revealed that both the model's test of residual heterogeneity and the moderator test were statistically significant. Concerning the various moderators, as with the previous analysis involving mice, the analysis identified year as a negative and significant influence on GCA variance. This result suggests that, as was found in the mice data, the proportion of variance explained by GCA has decreased among rats over about 90 years (Table 5). The analysis also identified a significant negative effect of task number (indicating that smaller numbers of tasks were associated with greater GCA variance), but no effect of factor estimation technique. Fig. 10 illustrates the decline of GCA variance in rats as a function of data collection year.

8. Discussion

The results of the cross-temporal meta-analyses yielded indications of substantial variance associated with GCA in both laboratory mice and Norwegian rats (32.2% and 54.5% respectively). This is comparable to values found in large meta-analytic studies of human populations (Warne & Burningham, 2019). It should be noted that these values may underestimate the true GCA saturation across tasks and across subjects if there are second-order correlations among factors, which cannot be accounted for using the current dataset (see Warne & Burningham, 2019 for similar arguments in the analysis of data on human GCA variance).

Temporal declines in proportion of GCA variance (consistent with a "differentiation effect") spanning approximately one century are evident in the case of both species. These declines are not confounded with age, sex, latent variable modeling method, or task number in the case of mice, or with task number or latent variable modeling method in the case of rats. The availability of fewer effect sizes and data on relevant moderators necessitates caution when interpreting temporal trends in the case of the latter however. There were no indications of missing studies based on the use of trim-and-fill in either species; but for mice, there were indications of significant funnel asymmetry, which might be attributable to three studies featuring large effect sizes and large standard errors.

In rats, task number was an independent significant negative predictor of GCA variance, meaning that GCA variance is greatest when task number is lowest. It has been noted that some studies from the "first wave" examining GCA in Norwegian rat populations, and other taxa, may have oversampled narrow domains, such as those associated with spatial reasoning and learning (Locurto, 1997). A good example of this is Thorndike's (1935) study, which evaluated rat performance with respect to 32 tasks, most of which measured aspects of spatial reasoning. Crinella and Yu (1995) have discussed this issue in relation to certain cognitive batteries that have been employed in rats during the "second wave," specifically the Thompson and Anderson Batteries, which (while employing fewer measures relative to, e.g., Thorndike, 1935) nevertheless attempt to tap a broader set of domains inclusive of visual, spatial, tactile, and possibly also olfactory ability. They argued that while these domains may not be especially diverse when considered in

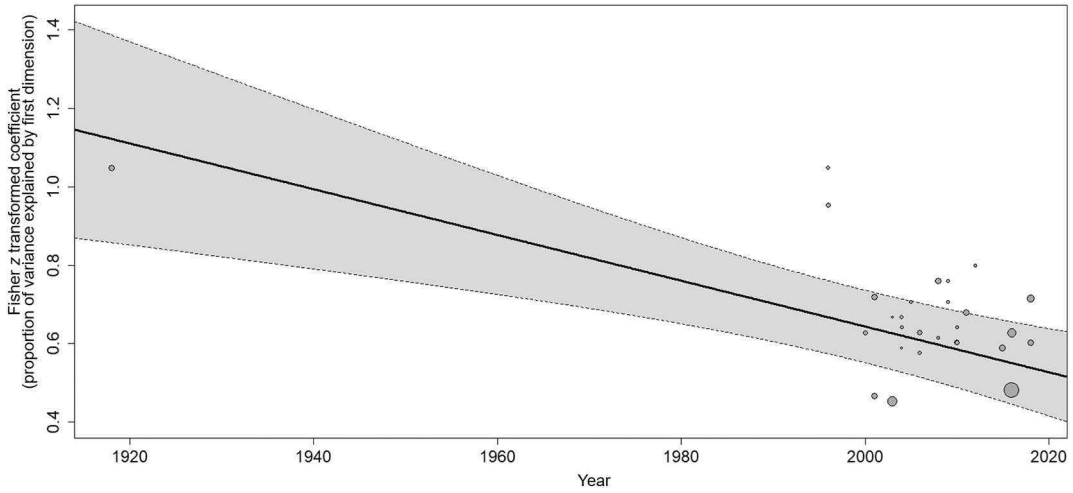

Fig. 6. Meta-regression evaluating the influence of data collection year on the *z*-transformed GCA variance coefficients across a sample of 28 mouse effect sizes.

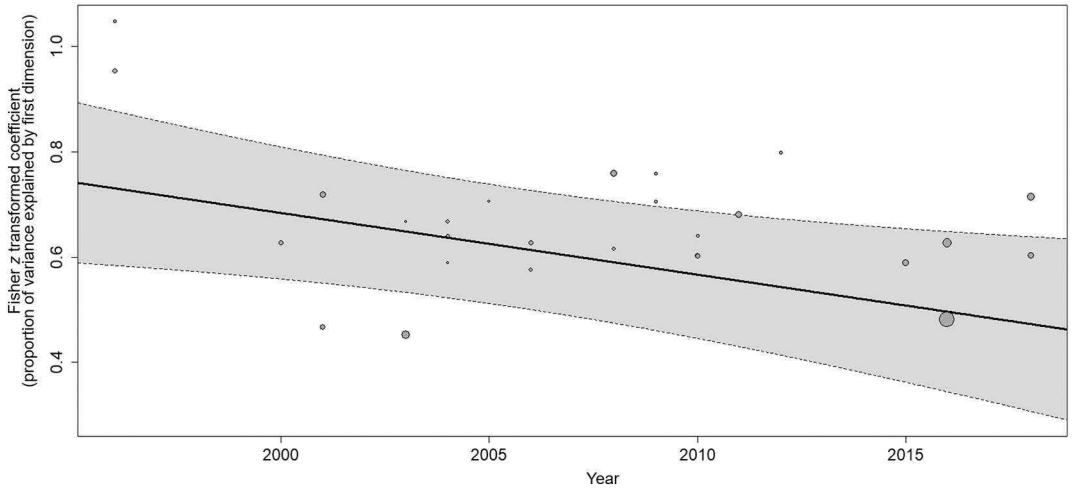

Fig. 7. Meta-regression evaluating the influence of data collection year on *z*-transformed GCA variance coefficients across a sample of 27 mouse effect sizes, excluding Bagg (1920).

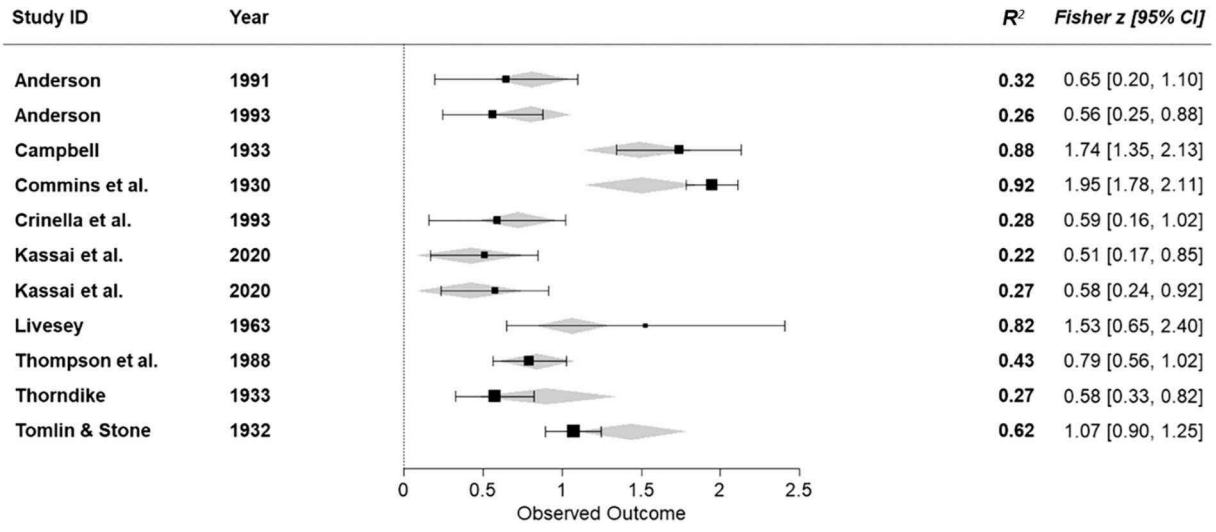

Fig. 8. Forest plot with *z*-transformed GCA coefficients across a sample of 11 GCA variance effect sizes in rats, along with data collection years. The model was generated with the *rma* function and the R package *metafor*.

Table 4
Results of the trim-and-fill test and the Egger's regression analysis of asymmetry involving 11 GCA variance effect sizes in rats.

Trim-and-Fill				
Studies trimmed	*Point estimate*	*95% CI*	*p-value*	*Q*
Observed values = 10	0.945	0.642, 1.249	$p < .0001$	178.432
Adjusted values = 10	0.945	0.642, 1.659	$p < .0001$	178.432
Regression Test for Funnel Asymmetry				
Estimate	*95% CI*	*t-value*	*Df*	*p-value*
1.668	0.691, 2.645	−1.41	9	0.1918

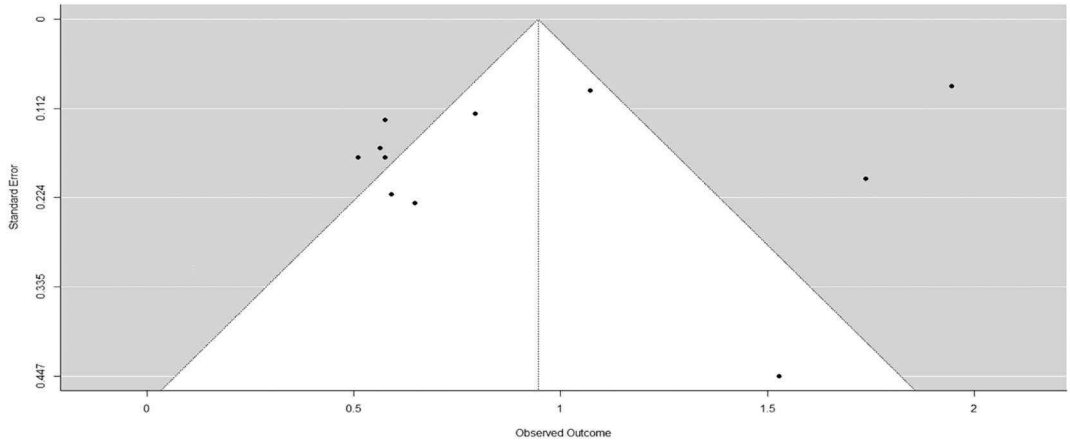

Fig. 9. Funnel asymmetry plot with trim-and-fill imputed "missing" studies (unfilled circles—in this case there were no missing studies) evaluating the absence of data points of *z*-transformed GCA variance coefficients across a sample of 11 effect sizes in rats. The graphic was generated with the R package *metafor*.

Table 5
Results of a multivariate cross-temporal meta-analysis model examining the moderating effects of number of tasks, latent variable modeling method, and data collection year on the proportion of variance explained by GCA across 11 rat effect sizes.

Multivariate Meta-Analysis Model (k = 11; method: ML)					
Test for Residual Heterogeneity:					
$QE(df = 7) = 44.307$, *p-value* $< .0001$					
Test of Moderators (coefficients 2:4):					
$QM(df = 3) = 26.534$, *p-value* $< .0001$					
Parameters (reference category)	*Estimate*	*95% CI*	*SE*	*z-value*	*p-value*
Intercept	25.108	(10.531, 39.685)	7.437	3.38	0.0007
Latent Variable Modeling Method (PAF)	−0.054	(−0.490, 0.383)	0.223	−0.24	0.8098
Number of tasks	−0.021	(−0.032, −0.009)	0.006	−3.54	0.0004
Year	−0.012	(−0.019, −0.005)	0.004	−3.27	0.0011
Fit indicator	***logLik***	***Deviance***	***AIC***	***BIC***	***AICc***
Value	0.763	17.917	8.475	10.464	20.475

light of Jensen's (1998) nomological breadth criterion for a "good GCA factor," this standard is likely easier to attain in human studies.

Crinella and Yu's (1995) observations furthermore suggest that caution should be exercised when interpreting the first dimension in rodents as GCA. Many researchers in this area of comparative psychology routinely and explicitly interpret this as GCA or "general intelligence" (e.g., Crawford et al., 2020; Galsworthy et al., 2002, 2005; Kolata et al., 2010; Light et al., 2010). However, the possibility also exists that, because of relatively low nomological breadth (relative to human batteries), such a factor might correspond more closely to a narrower group factor, perhaps something more akin to domain-general spatial reasoning or learning.[6] Whether or not Spearman's (1927) principle of the indifference of the indicator (with respect to GCA) holds in non-human animal subjects is yet to be adequately tested. A related debate concerns whether or not apparent GCA factors derived from one species are fully translatable between species (one might ask, "do species exhibit compositionally distinct GCA factors reflecting their unique adaptive histories, or is it the same GCA, just expressed to different degrees between species?"; see Burkart et al., 2017). Some recent data indicate that GCAs are (to a limited degree) translatable between different species (of, e.g., primates) evaluated using a common behavioral-psychometric assessment framework (for different approaches to addressing this issue, see Kaufman, Reynolds, & Kaufman, 2019; Woodley of Menie & Peñaherrera-Aguirre, 2022).

Crinella and Yu (1995) argued that the apparently weak GCA factor in their study of unlesioned rats, relative to Anderson's (1993, 1995) findings of such a factor, may in part be a function of the use of different strains (specifically Long-Evans vs. Sprague-Dawley), controlling for strain (by modeling it as a random effect) did not eliminate heterogeneity in GCA variance in either the mouse or rat experimental populations. Moreover, no direct evidence was found for a significant difference between these two strains when GCA variance was (re) extracted from the relevant correlation matrices (see Fig. 7).[7]

An interesting observation made by Crinella and Yu (1995) is that GCA variance might present to a substantially greater degree among lesioned rats (relative to unlesioned ones). This finding strengthens the core hypothesis motivating the current analyses, specifically that improvements in environmental factors, in particular those related to

[6] Nicholas Mackintosh (1935–2015) publicly expressed a similar view while being interviewed for the Distinguished Contributor Award at ISIR in 2013.

[7] It should be noted that in re-estimating Anderson's (1993) rat GCA factor loadings, they excluded one of his four measures on the basis of non-independence.

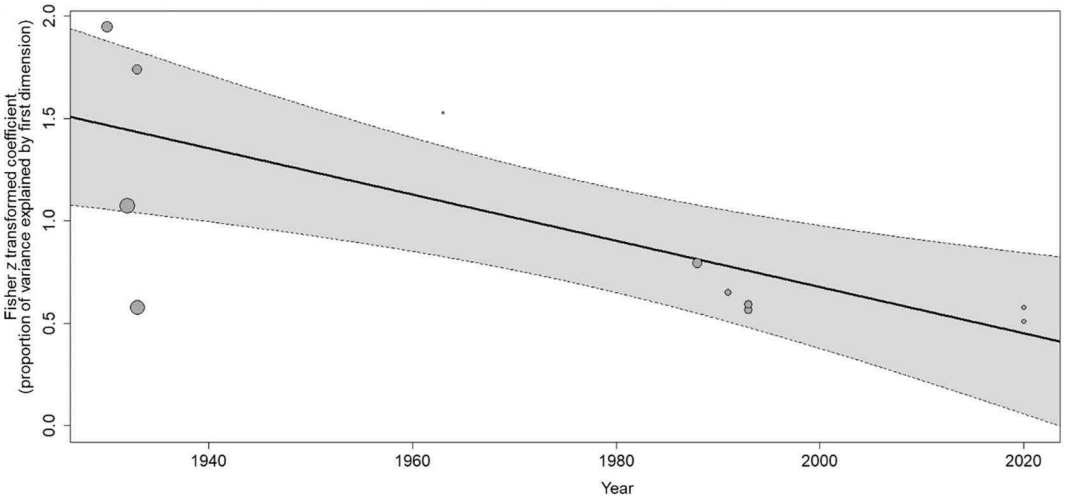

Fig. 10. Meta-regression evaluating the influence of data collection year on *z*-transformed GCA variance coefficients across a sample of 11 rat effects sizes.

microbiological quality and organismal health, may be a powerful driving force behind the trend toward greater cognitive differentiation among laboratory rodent populations. This is because negative cognitive effects from lesioning might resemble the broad effects of forms of environmental stress and associated organic damage (such as that caused by microbial infections), which, via their impacts on central nervous system integrity, might handicap the phenotypic expression of specific cognitive processes, drawing them into correlation with one another to a greater degree historically (when the influences of such adverse environments were more potent). Moreover, this is consistent with the LH model, as more strongly intercorrelated abilities are likely to be an adaptive *condition-dependent* response to the presence of heightened environmental risk. This is because cognitive generalism (as evidenced by the presence of stronger GCA covariance) is thought to prepare organisms for unpredictable and harsh regimes of environmental risk, by enhancing their capacity for contingent switching between unstable environmental niches (Woodley et al., 2013).

This strengthens the inference to the idea that an actual Flynn effect is present in these data, as rodents that are in poorer condition (whether as a result of passive exposure to heightened environmental risk, or due to the experimental induction of forms of brain damage) might reasonably be expected to exhibit lower performance means (in addition to generally greater inter-individual variance with respect to cognitive performance), relative to "healthy" rodents. Congruently, Sauce et al. (2018) noted that when mice were exposed to highly cognitively enriched environments, cognitive performance levels were greatly elevated (by the equivalent of 6.6 "IQ points"), relative to those found in the control population. These researchers also observed that heritability appeared to decline in tandem with the ability gain (consistent with reduced inter-individual cognitive variance and possibly also covariance).

As mentioned in the introduction, the LH model can be directly tested experimentally in laboratory rodents via measurement of the strength of correlations among abilities in addition to learning potential in different populations exposed to different levels of environmental risk. There may also be other trends accompanying the ones noted in the current analysis that might be expected were LH characteristics changing in more recent laboratory populations of these rodents. For example, there may be increases in brain mass in addition to other potential outcomes of increased somatic effort. One set of findings potentially consistent with this are those of Klimentidis et al. (2011), who examined trends in body weight in a sample of more than 20,000 animals covering eight species. These included laboratory mice and both captive and feral (urban and rural) Norwegian rats. Increases in body weight were noted in all cases. Among the various prospective causes of these trends, Klimentidis et al. (2011) note that "[o]ther explanations may include epigenetic-mediated programming of growth and energy-allocation patterns owing to any number of environmental cues such as stressors, resource availability, release from predation or climate change" (p. 1631). As some of these prospective causes might be associated with *reductions* in environmental risk, these positive body weight trends may (in part) be a consequence of (possibly miscalibrated) epigenetically mediated increases in somatic effort stemming from LH slowing in these rodent (and other) populations.

Another (but possibly complementary) hypothesis, which cannot be ruled out based on the current results, is that the "differentiation effect" noted here stems from the action of genetic changes occurring in these populations of laboratory rodents over time. One possibility is that laboratory rodents used in behavioral research might have been bred for enhanced competence with respect to narrower domains of cognition, reflecting the shift away from individual differences research that started in the early 1940s (the start of the "dark age"). Indeed, this may even have been to an extent a "self-fulfilling prophecy," in that the experimental focus on performance with respect to very narrow learning domains or to conditioning during this period, without regard to individual differences or large factors underlying them, may have (inadvertently) led to the selective propagation of strains in which GCA variance was attenuated, yielding laboratory populations that were more amenable to working within the constraints of the behavioristic experimental paradigm more broadly. It is conceivable that this hypothetical selection may have acted specifically so as to favor increased cognitive plasticity via enhancement of the capacity to form secondary modules, which underlie the ability to acquire narrow, environmentally sensitive abilities through the application of domain-general learning mechanisms (akin to "muscle memory" acquired in the course of learning to play a computer game, or musical instrument; for discussion of these, see Burkart et al., 2017; Woodley of Menie, Peñaherrera-Aguirre, & Jurgensen, 2022). Rodents that are better able to develop these secondary modules through exposure to repeated stimuli might be expected to have weaker GCA manifolds. This hypothesized genetic preparedness for cognitive plasticity might also have provided a genetic substrate on which the Flynn effect could act to a greater extent among more recently bred rodent populations, with more innately cognitively plastic populations being better able to engage in the sorts of LH tradeoffs necessary for the cultivation of at least some subset of narrow abilities, relative to their less genetically plastic ancestors. Another possibility is that more recent strains are more inbred. The resultant loss of genetic and phenotypic diversity might be expected to attenuate the strength of phenotypic correlations among ability measures, although the use of strain as a random effect in the current analyses might have gone some way toward

having controlled for this potential confound, as heterogeneity in the degree of inbreeding among the various strains considered would have been (indirectly) controlled in these analyses.

It should be stressed that as the confounds discussed above could not be addressed in the current study, and as changes in mean performance over time could not be directly established among these populations, the results of this analysis, while certainly suggestive of the presence of the Flynn effect among laboratory rodent populations, are nevertheless speculative. Early in the exploration of novel scientific topics more speculative findings can catalyze more rigorous research. Ultimately, and to this end, in order to comprehensively test for Flynn effects in rodent populations, experimental designs along the lines discussed in the introduction would be ideal. Such efforts aimed at specifically testing the long-term influences of a variety of prospective causes and meta-causes of the Flynn effect (or its reversal) could help to solve many of the enduring mysteries surrounding this enigmatic effect.

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Data availability

All data are made available in tabulated form within the manuscript

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