Careers in Clinical Academia

Academy of Medical Sciences Report

Decline in Clinical Academics

Data from the Medical Schools Council (MSC) Clinical Academic Survey show a 4% decline in clinical academic numbers over the last decade (positions at professor, senior lecturer, and lecturer level). Further analysis of these figures reveals that this decline is particularly acute at the mid-career level (senior lecturer), where there has been a 25% decline in numbers across the UK. Despite increases at more senior levels (professorship), when clinical academics at consultant level are expressed as a proportion of the whole consultant workforce, we can see a steady decline from 8.55% in 2011 to 5.7% in 2020. The proportion of clinical academic GPs in England has remained stubbornly low with just between 0.6% and 0.7% of total numbers of GPs over the same period.298

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Professorships in child and adolescent psychiatry relative to a similarly sized medical specialty in the UK and Ireland: cross-sectional study

Ian Kelleher, Aleksandra Z. Poziemska, Valentina Kieseppä, Anita Thapar, Bernadka Dubicka, Elaine Lockhart, Tamsin Ford, Helen Minnis, Louise Gallagher, Fiona McNicholas and Kirstie O'Hare

Background

A youth mental health crisis is considered one of the great challenges of our time, and research and clinical services in child and adolescent psychiatry have become a priority for governments and funders. Academic leadership is needed to drive forward research. It is not clear how many senior academic leadership posts (professorships) there are in child and adolescent psychiatry, nor how this benchmarks against a similarly sized medical specialty.

Aims

This study aimed to determine the number of professorships in child and adolescent psychiatry in the UK and Ireland compared to a similarly sized specialty. A secondary aim was to identify the number of clinical trials registered for mental and behavioural disorders in children.

Method

We identified registered specialists in child and adolescent psychiatry and a similarly sized specialty who held full professorships in medical schools. We searched the International Standard Randomised Controlled Trial Number (ISRCTN) and ClinicalTrials.gov for trials.

Results

As of 23 March 2023, there were 1725 doctors on the General Medical Council's (GMC) specialist register in child and adolescent psychiatry. The closest specialty in terms of number of registered specialists was neurology ($N = 1724$). We identified 24 professors in child and adolescent psychiatry across the UK and Ireland, compared to 124 in neurology. For every intervention trial registered for mental and behavioural disorders in children, there were approximately ten trials registered for diseases of the nervous system.

Conclusions

Despite equivalent numbers of medical specialists in child and adolescent psychiatry and neurology, there is a striking disparity in the number of professorship appointments. While young peoples' mental health has, ostensibly, become a priority for policy-makers and funders, this is not reflected in medical professorship appointments. The paucity of senior academic child and adolescent psychiatrists has real-world implications for training, research, innovation and service development in mental health services.

Keywords

Academic psychiatry; child and adolescent psychiatry.

Table 1 Number of professorships in child and adolescent psychiatry and neurology in Ireland and the UK (with breakdown for the four nations of the UK)

	Professors in neurology	Professors in child and adolescent psychiatry
Ireland	9	2
England	102	17
Scotland	9	4
Wales	3	1
Northern Ireland	1	0
Total UK	115	22
Total UK and Ireland	124	24

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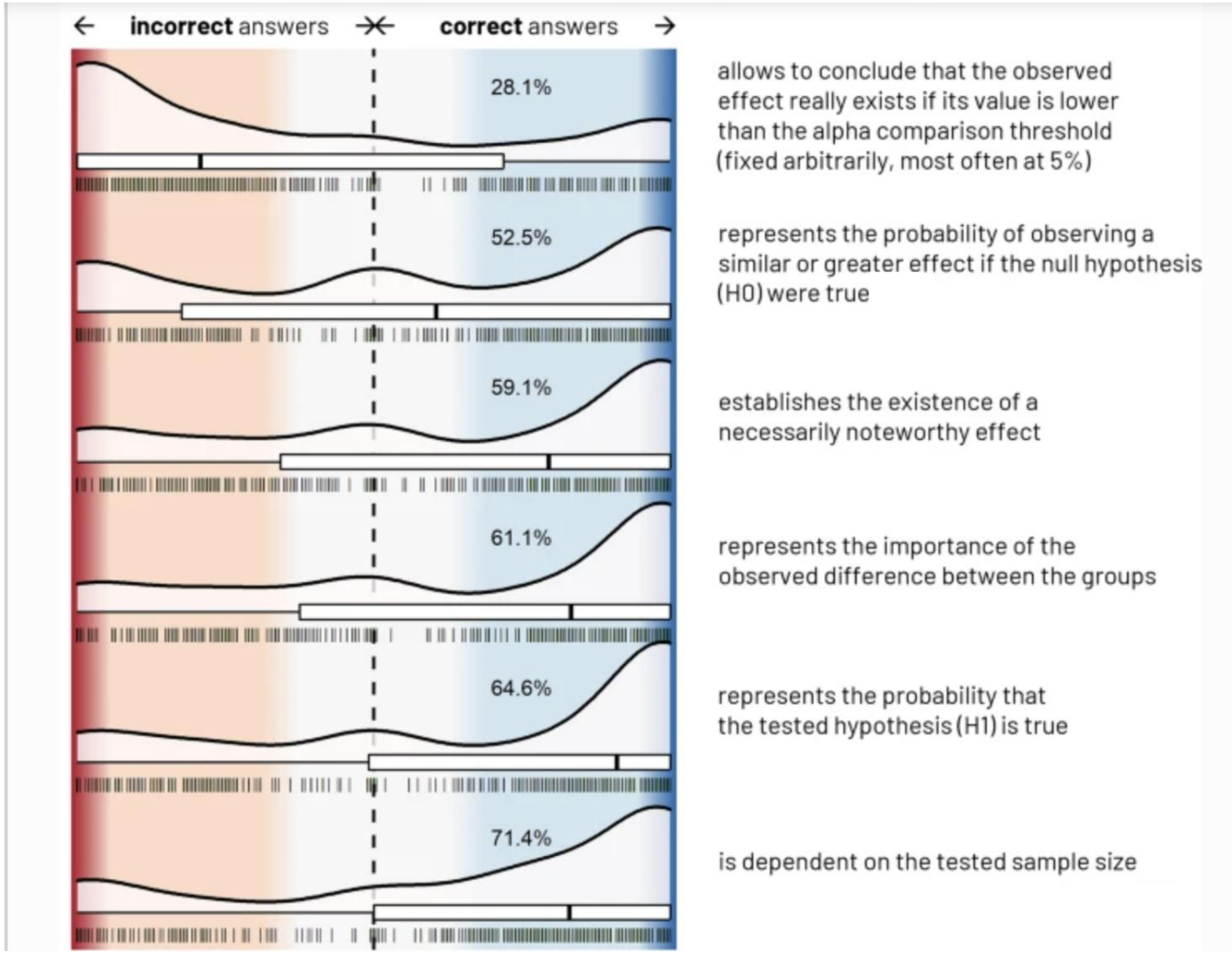

Original article | Open access | Published: 20 April 2023

Illusion of knowledge in statistics among clinicians: evaluating the alignment between objective accuracy and subjective confidence, an online survey

Camille Lakhlifi, François-Xavier Lejeune, Marion Rouault, Mehdi Khamassi & Benjamin Rohaut

Cognitive Research: Principles and Implications **8**, Article number: 23 (2023) | Cite this article

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Apart from the structural factors mentioned above, consider this:

Ability to wait for uncertain rewards.

Clinical work evolves at fast time scales and is nearly always rewarding.

Clinical work finishes and you can switch off

Research work can be on your mind all the time

Research work evolves over long time scales and has uncertain outcomes.

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In research work you are constantly under evaluation: you are only as good as your last grant.

In clinical work you are held to high standards, but your position, team etc are not (or should not be) at peril.

In clinical work, you can go to work even if you do not have an inspiration, a new Idea etc

In research work, you will struggle to be generative, to pursue etc.

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Failure is the norm in research work: ~ 70% of my grant applications failed.

Failure is the norm in research work: all of my papers have required revisions,

Failure is the norm in research work: ~ 70% of my papers were first rejected.

Negative peer evaluation is common: “this is the worst paper I have ever read”

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Research is creative

Research stays around: it is something you generated and will last

Research leads to (constant) innovation

Research allows you to question things (everything)

Research means you meet lots of very smart people

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Acta Neuropathol (1997) 93 : 215–218

EXPRESS COMMUNICATION

Argyris K. Stringaris · Wolfgang Brück
Hayrettin Tumani · Holger Schmidt · Roland Nau

Increased glutamine synthetase immunoreactivity in experimental pneumococcal meningitis

Received: 23 September 1996 / Revised, accepted: 21 November 1996

Abstract Glutamine synthetase (GS), glial fibrillary acidic protein (GFAP) immunohistochemistry and neuronal apoptotic cell death were evaluated in a rabbit model of pneumococcal meningitis. Meningitis caused an increase of GS immunoreactivity in the cerebral cortex, but not in the hippocampal formation. GFAP immunoreactivity remained unchanged. This may represent a protective mechanism for cortical neurons. The inability of hippocampal GS to counteract the detrimental effects of glutamate may be the cause of neuronal apoptosis observed in the dentate gyrus during meningitis.

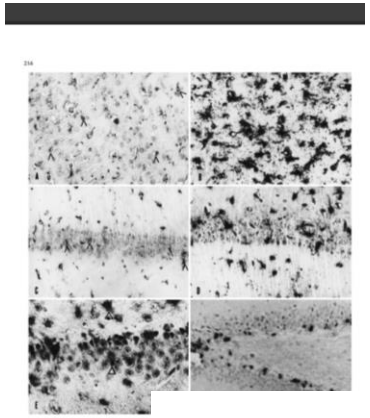
Key words Glutamine synthetase · Immunohistochemistry · Meningitis · *Streptococcus pneumoniae* · Apoptosis

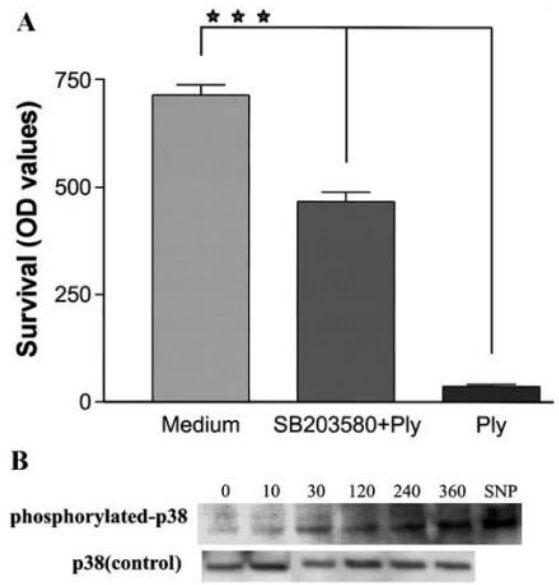
astrocytes, is implicated in glutamate detoxification by converting it to glutamine [2, 8]. For this reason, we evaluated GS expression by immunohistochemistry in the hippocampal formation and cerebral cortex in the rabbit model of experimental pneumococcal meningitis.

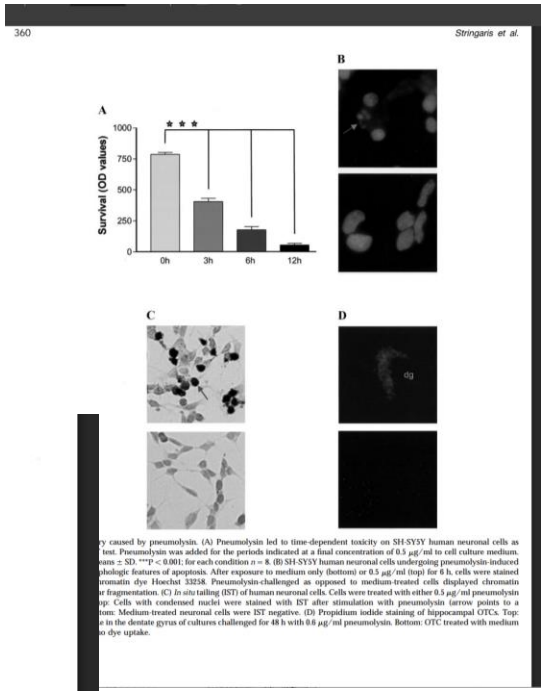
Materials and methods

Experimental protocol

A *Streptococcus pneumoniae* type 3 strain originally isolated from an adult with meningitis was used. After several passages in rabbits, infected cerebrospinal fluid was cultured on blood agar plates and bacteria were suspended in sterile saline solution. Anesthesia was induced by intramuscular injections of ketamine (25 mg/kg) and xylazine (5 mg/kg) and maintained with intravenous urethane







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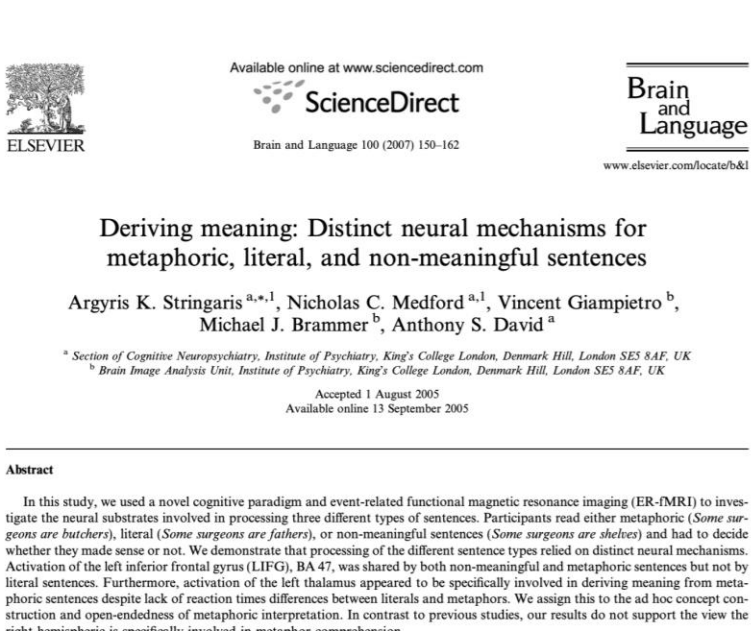

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Deriving meaning: Distinct neural mechanisms for metaphoric, literal, and non-meaningful sentences

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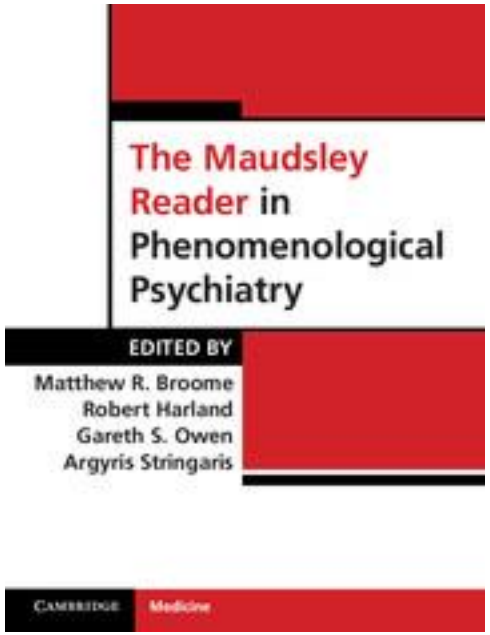
Accepted 1 August 2005
Available online 13 September 2005

Abstract

In this study, we used a novel cognitive paradigm and event-related functional magnetic resonance imaging (ER-fMRI) to investigate the neural substrates involved in processing three different types of sentences. Participants read either metaphoric (*Some surgeons are butchers*), literal (*Some surgeons are fathers*), or non-meaningful sentences (*Some surgeons are shelves*) and had to decide whether they made sense or not. We demonstrate that processing of the different sentence types relied on distinct neural mechanisms. Activation of the left inferior frontal gyrus (LIFG), BA 47, was shared by both non-meaningful and metaphoric sentences but not by literal sentences. Furthermore, activation of the left thalamus appeared to be specifically involved in deriving meaning from metaphoric sentences despite lack of reaction times differences between literals and metaphors. We assign this to the ad hoc concept construction and open-endedness of metaphoric interpretation. In contrast to previous studies, our results do not support the view the



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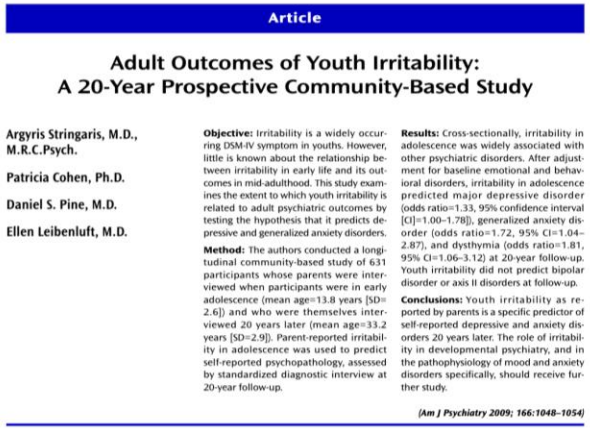


Article

Adult Outcomes of Youth Irritability: A 20-Year Prospective Community-Based Study

Argyris Stringaris, M.D., M.R.C.Psych.
Patricia Cohen, Ph.D.
Daniel S. Pine, M.D.
Ellen Leibenluft, M.D.

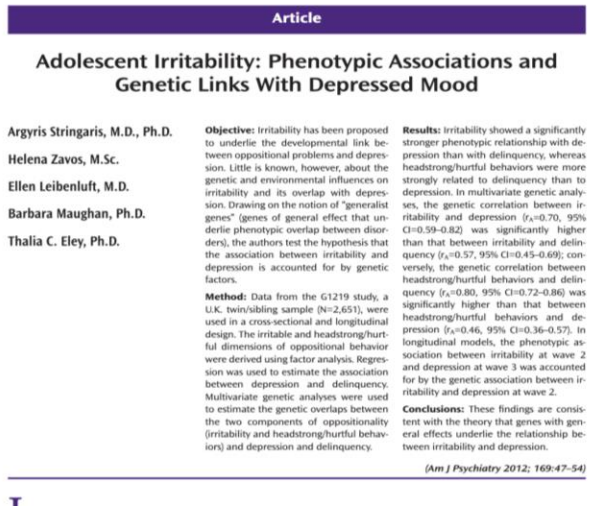
Objective: Irritability is a widely occurring DSM-IV symptom in youths. However, little is known about the relationship between irritability in early life and its outcomes in mid-adulthood. This study examines the extent to which youth irritability is related to adult psychiatric outcomes by testing the hypothesis that it predicts depressive and generalized anxiety disorders.

Method: The authors conducted a longitudinal community-based study of 631 participants whose parents were interviewed when participants were in early adolescence (mean age=13.8 years [SD=2.6]) and who were themselves interviewed 20 years later (mean age=33.2 years [SD=2.9]). Parent-reported irritability in adolescence was used to predict self-reported psychopathology, assessed by standardized diagnostic interview at 20-year follow-up.

Results: Cross-sectionally, irritability in adolescence was widely associated with other psychiatric disorders. After adjustment for baseline emotional and behavioral disorders, irritability in adolescence predicted major depressive disorder (odds ratio=1.33, 95% confidence interval [CI]=1.00–1.78), generalized anxiety disorder (odds ratio=1.72, 95% CI=1.04–2.87), and dysthymia (odds ratio=1.81, 95% CI=1.06–3.12) at 20-year follow-up. Youth irritability did not predict bipolar disorder or axis II disorders at follow-up.

Conclusions: Youth irritability as reported by parents is a specific predictor of self-reported depressive and anxiety disorders 20 years later. The role of irritability in developmental psychiatry, and in the pathophysiology of mood and anxiety disorders specifically, should receive further study.

(Am J Psychiatry 2009; 166:1048–1054)



Article

Adolescent Irritability: Phenotypic Associations and Genetic Links With Depressed Mood

Argyris Stringaris, M.D., Ph.D.
Helena Zavos, M.Sc.
Ellen Leibenluft, M.D.
Barbara Maughan, Ph.D.
Thalia C. Eley, Ph.D.

Objective: Irritability has been proposed to underlie the developmental link between oppositional problems and depression. Little is known, however, about the genetic and environmental influences on irritability and its overlap with depression. Drawing on the notion of "generalist genes" (genes of general effect that underlie phenotypic overlap between disorders), the authors test the hypothesis that the association between irritability and depression is accounted for by genetic factors.

Method: Data from the G1219 study, a U.K. twin/sibling sample (N=2,651), were used in a cross-sectional and longitudinal design. The irritable and headstrong/hurtful dimensions of oppositional behavior were derived using factor analysis. Regression was used to estimate the association between depression and delinquency. Multivariate genetic analyses were used to estimate the genetic overlaps between the two components of oppositionality (irritability and headstrong/hurtful behaviors) and depression and delinquency.

Results: Irritability showed a significantly stronger phenotypic relationship with depression than with delinquency, whereas headstrong/hurtful behaviors were more strongly related to delinquency than to depression. In multivariate genetic analyses, the genetic correlation between irritability and depression (r_A=0.70, 95% CI=0.59–0.82) was significantly higher than that between irritability and delinquency (r_A=0.57, 95% CI=0.45–0.69); conversely, the genetic correlation between headstrong/hurtful behaviors and delinquency (r_A=0.80, 95% CI=0.72–0.86) was significantly higher than that between headstrong/hurtful behaviors and depression (r_A=0.46, 95% CI=0.36–0.57). In longitudinal models, the phenotypic association between irritability at wave 2 and depression at wave 3 was accounted for by the genetic association between irritability and depression at wave 2.

Conclusions: These findings are consistent with the theory that genes with general effects underlie the relationship between irritability and depression.

(Am J Psychiatry 2012; 169:47–54)

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nature human behaviour

Article

https://doi.org/10.1038/s41562-023-01519-7

A highly replicable decline in mood during rest and simple tasks

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Check for updates

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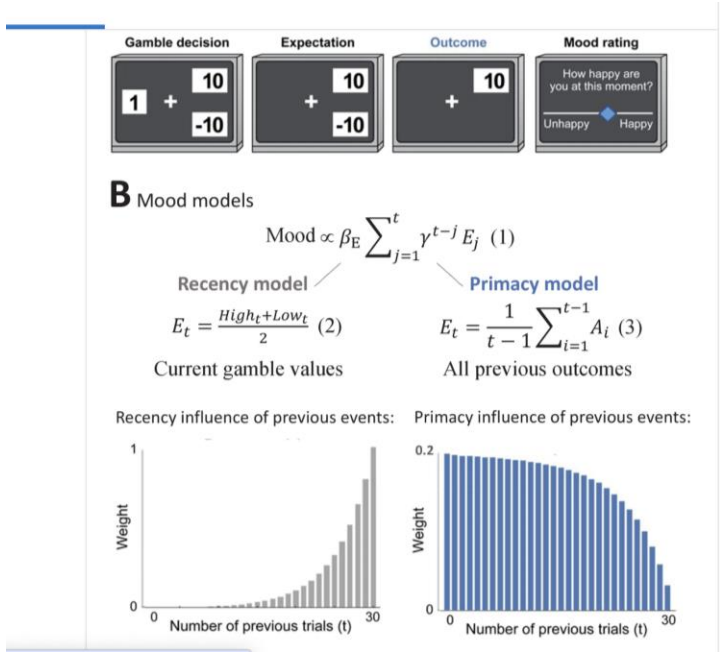
Does our mood change as time passes? This question is central to behavioural and affective science, yet it remains largely unexamined. To investigate, we intermixed subjective momentary mood ratings into repetitive psychology paradigms. Here we demonstrate that task and rest periods lowered participants' mood, an effect we call 'Mood Drift Over Time'. This finding was replicated in 19 cohorts totalling 28,482 adult and adolescent participants. The drift was relatively large (−13.8% after 7.3 min of rest, Cohen's $d = 0.574$) and was consistent across cohorts. Behaviour was also impacted: participants were less likely to gamble in a task that followed a rest period. Importantly, the drift slope was inversely related to reward sensitivity. We show that accounting for time using a linear term significantly improves the fit of a computational model of mood. Our work



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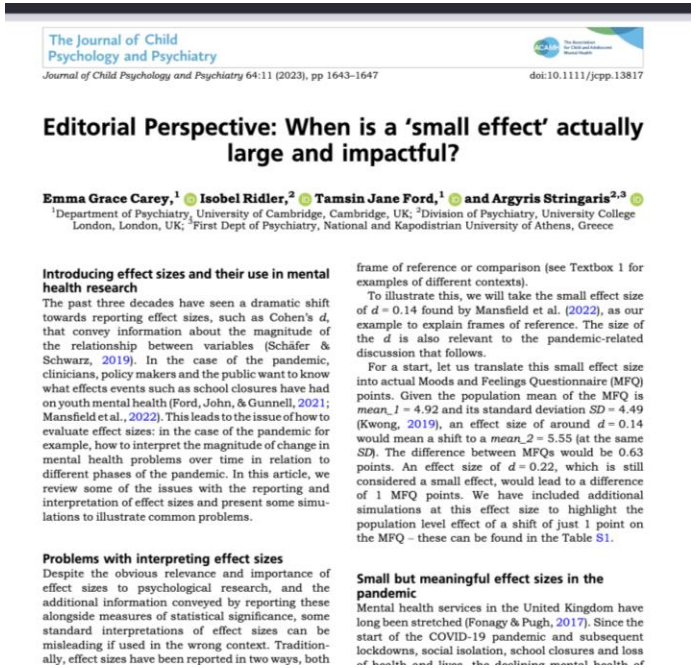
Open access Systematic review

BMJ Mental Health

Comparing apples and oranges in youth depression treatments? A quantitative critique of the evidence base and guidelines

Argyris Stringaris ,[1,2] Charlotte Burman,[1] Raphaelle Delpech ,[1] Rudolph Uher,[3] Dayna Bhudia ,[1] Despoina Miliou,[2] Ioannis-Marios Rokas ,[2] Marinos Kyriakopoulos ,[2,4,5] Lucy Foulkes ,[6] Carmen Moreno ,[7,8] Samuele Cortese ,[9] Glyn Lewis ,[1] Georgina Krebs [1]



The Journal of Child Psychology and Psychiatry

Journal of Child Psychology and Psychiatry 64:11 (2023), pp 1643–1647 doi:10.1111/jcpp.13817

Editorial Perspective: When is a 'small effect' actually large and impactful?

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Introducing effect sizes and their use in mental health research

The past three decades have seen a dramatic shift towards reporting effect sizes, such as Cohen's *d*, that convey information about the magnitude of the relationship between variables (Schäfer & Schwarz, 2019). In the case of the pandemic, clinicians, policy makers and the public want to know what effects events such as school closures have had on youth mental health (Ford, John, & Gunnell, 2021; Mansfield et al., 2022). This leads to the issue of how to evaluate effect sizes: in the case of the pandemic for example, how to interpret the magnitude of change in mental health problems over time in relation to different phases of the pandemic. In this article, we review some of the issues with the reporting and interpretation of effect sizes and present some simulations to illustrate common problems.

Problems with interpreting effect sizes

Despite the obvious relevance and importance of effect sizes to psychological research, and the additional information conveyed by reporting these alongside measures of statistical significance, some standard interpretations of effect sizes can be misleading if used in the wrong context. Traditionally, effect sizes have been reported in two ways, both frame of reference or comparison (see Textbox 1 for examples of different contexts).

To illustrate this, we will take the small effect size of $d = 0.14$ found by Mansfield et al. (2022), as our example to explain frames of reference. The size of the *d* is also relevant to the pandemic-related discussion that follows.

For a start, let us translate this small effect size into actual Moods and Feelings Questionnaire (MFQ) points. Given the population mean of the MFQ is $mean\_1 = 4.92$ and its standard deviation $SD = 4.49$ (Kwong, 2019), an effect size of around $d = 0.14$ would mean a shift to a $mean\_2 = 5.55$ (at the same *SD*). The difference between MFQs would be 0.63 points. An effect size of $d = 0.22$, which is still considered a small effect, would lead to a difference of 1 MFQ points. We have included additional simulations at this effect size to highlight the population level effect of a shift of just 1 point on the MFQ – these can be found in the Table S1.

Small but meaningful effect sizes in the pandemic

Mental health services in the United Kingdom have long been stretched (Fonagy & Pugh, 2017). Since the start of the COVID-19 pandemic and subsequent lockdowns, social isolation, school closures and loss

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Institute of Psychiatry MRCPsych Intake October 2002

Back Row - Drs J. Twaddle, G. Wilson, A. Stringaris, T. Lavender, N. Weir, D. Okai, N. Harrison, C. McCurrie
Front Row: Drs K. Bailey, C. Commane, C. Penny, A. Mbamali, A. Raznahan, E. Langan, J. Das-Munshi, E. Chu

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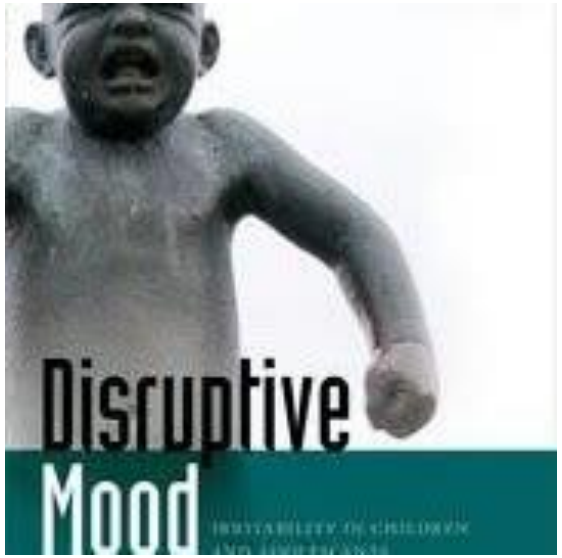

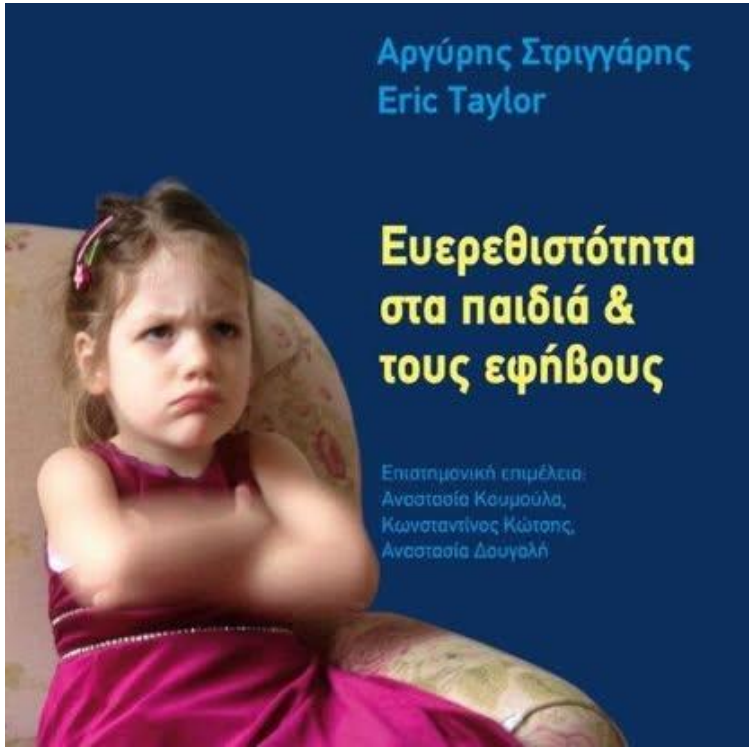

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