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On the Prevalence of Congenital Amusia

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Congenital amusia, or 'tone deafness,' is a lifelong impairment in musical ability, reported to be present in approximately 4% of the general population. We examined the meaningfulness of 4% as an estimate of the prevalence of amusia given current test-based methods; here we focused on the Distorted Tunes Test (DTT) and the Montreal Battery of Evaluation of Amusia (MBEA). We demonstrate that estimates of prevalence critically depend on the specific cutoff applied to the test and the degree of skew in the distribution of scores. Broader consideration of this issue reveals that the use of arbitrary cutoffs is not unique to diagnosis of congenital amusia. We conclude that although the MBEA has shown to be a valuable diagnostic tool, caution is warranted against attributing meaning to the reported 4% rate of congenital amusia that is so widely cited in the literature.

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Key words: amusia, Distorted Tunes Test, MBEA, musical ability, tone deafness

Congenital amusia, or 'tone-deafness,' refers to lifelong impairment in musical ability that is unrelated to hearing acuity, general neurological functioning, or exposure to music (Ayotte, Peretz, & Hyde, 2002). Musical impairments associated with congenital amusia have been linked primarily to pitch processing, with amusic individuals typically unable to detect small changes in pitch (Hyde & Peretz, 2004) or recognize melodies without the aid of lyrics (Ayotte et al., 2002). In the past ten years, there has been growing interest in congenital amusia, arguably driven by better appreciation of the relationship between music and language processing (Patel, 2008) and evidence that tone-deaf individuals also show impairments in visuospatial abilities (Douglas & Bilkey, 2007).

Given possible links between congenital amusia and aspects of language and spatial processing, the issue of the prevalence of congenital amusia in the population has attracted substantial attention. Since 2000, at least 20 papers have identified 4% as the percentage of the general population with congenital amusia. Sixteen of these have cited a single study, Kalmus and Fry (1980), as the source for this number (Ayotte et al., 2002; Cuddy, Balkwill, Peretz, & Holden, 2005; Foxton, Dean, Gee, Peretz, & Griffiths, 2004; Foxton, Nandy, & Griffiths, 2006; Hyde et al., 2007; Hyde & Peretz, 2004; Hyde, Zatorre, Griffiths, Lerch, & Peretz, 2006; Mandell, Schulze, & Schlaug, 2007; Peretz, 2006, 2008; Peretz, Brattico, Jarvenpaa, & Tervaniemi, 2009; Peretz, Brattico, & Tervaniemi, 2005; Peretz et al., 2008; Peretz & Hyde, 2003; Särkämö et al., 2009; Sloboda, Wise, & Peretz, 2005). Four additional studies cite 4%, but either do not provide a source or refer indirectly to the work of Kalmus and Fry (Douglas & Bilkey, 2007; Drayna, Manichaikul, de Lange, Snieder, & Spector, 2001; Patel, Foxton, & Griffiths, 2005; Thompson, 2007). The large number of studies reporting that 4% of the general population suffers from congenital amusia based on the results of a single study motivated us to take a closer look at: (1) methods used to assess prevalence, and (2) what estimates of prevalence using these methods mean.

In the widely cited Kalmus and Fry (1980) study, individuals completed a distorted tunes test (DTT) in which they listened to popular melodies with the task of detecting pitch errors. For an initial comparison of 'musical' participants (a BBC listening panel) and self-proclaimed tone-deaf participants, the authors determined by visual inspection that all participants in the 'musical' group made two or fewer misses and that three or more misses seemed to reliably distinguish the two groups. Applying the three-miss criterion to a large unselected sample ($n = 604$), the authors estimated that the prevalence of tone deafness in the population was around 4%. The rather arbitrary nature of this cutoff applied to a test without well-established psychometric properties suggests that there is reason to question how informative 4% is as a prevalence rate for congenital amusia (Ayotte et al., 2002; Hyde & Peretz, 2004).

Recently, the tool almost exclusively used to assess congenital amusia is a theoretically motivated set of tests of musical ability developed by Peretz and colleagues in part to address some of the weaknesses of the DTT

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(Peretz, Champod, & Hyde, 2003). The Montreal Battery of Evaluation of Amusia (MBEA) has six subtests, which assess melodic organization (Scale, Contour, Interval), temporal organization (Rhythm, Meter), and memory. Individual scores are averaged across subtests to produce a composite score that demonstrates a number of favorable psychometric properties including sensitivity, approximate normality, test-rest reliability, and convergent validity. Peretz and colleagues have argued that the MBEA confers a number of advantages for diagnosis of congenital amusia, including its specific use as a method to assess prevalence (Peretz et al., 2003, p. 68). Individuals are classified as tone deaf if their composite MBEA score is more than two standard deviations (*SD*s) below the mean. Based on this operational definition, the MBEA suffers the same arbitrary cut-off problem as Kalmus and Fry's (1980) method of assessing prevalence. The reason is that if the distribution of composite MBEA scores is normally distributed, as claimed, then a > 2 *SD* cutoff necessarily yields a population occurrence rate of 2.28% (Gravetter & Wallnau, 2006); see Figure 1a. If the distribution of scores is not normal, but negatively skewed, then the proportion of the general population estimated to be afflicted with congenital amusia will increase with increasing skew.

Figure 1b provides a concrete illustration of how estimated prevalence of congenital amusia varies with skew. We generated a family of epsilon-skew-normal distributions (Mudholkar & Hutson, 2000) with varying amounts of skew, ε, and mode equal to the mode of published MBEA norms.[1] Next, we determined the expected percentage of individuals to be afflicted with congenital amusia for each distribution. The value of the skew parameter, ε, which best fit the published norms was 0.17, yielding a population value of ~3.2%.; this value matches the 3.2% estimate of prevalence determined by applying the 2 *SD* cut off to the published MBEA norms. Estimated prevalence is increased to the reported 4% (Kalmus & Fry, 1980) by modestly adjusting the skew parameter, ε, to 0.26. The results of these simulations show that based on the 2 *SD* cut off used with the MBEA, a 4% percentage value for the prevalence of congenital amusia provides no more information than the degree of negative skew in an otherwise normal distribution of scores.

One natural question that emerges from this analysis concerns how widespread the problem is. That is, does it extend to other disorders? To address this question, we examined methods used to assess the prevalence of several other disorders, including dyslexia, dyscalculia, and developmental prosopagnosia. Methods for assessing learning disabilities, such as dyslexia and dyscalculia, have traditionally relied on a discrepancy between achievement (reading or mathematics achievement, respectively), as assessed by standardized tests, and intellectual ability, as assessed by IQ score on the Wechsler Intelligence Scale for Children (WISC) or Wechsler Adult Intelligence Scale (WAIS). For example, a commonly used method to diagnose dyslexia and dyscalculia requires that an individual fall below an achievement criterion on the ability of interest (e.g., > 2 *SD* below the mean) and above an IQ criterion (e.g., > 90) (Lewis, Hitch, & Walker, 1994; Lindgren, de Renzi, & Richman, 1985; Vicari et al., 2005). A modification of this technique involves predicting achievement from IQ or age using linear regression, and then applying a cutoff to the predicted achievement scores (Dykman & Ackerman, 1992; Gross-Tsur, Manor, & Shalev, 1996; Lindgren et al., 1985). In general, tests for assessing achievement and ability vary widely, and cutoffs are applied inconsistently (Forness, Sinclair, & Guthrie, 1983; Shalev, Auerbach, Manor, & Gross-Tsur, 2000). In this respect, it is not surprising that reported rates of dyslexia vary widely, from around 3% to 6% (Ramus, 2003; Shalev et al., 2000; Shalev & Gross-Tsur, 2001; Vicari et al., 2005) to as high as 17.5% (Démonet, Taylor, & Chaix, 2004; Shaywitz, 1998).

Diagnosis of developmental prosopagnosia is generally based on poor performance on one of several standardized batteries of face processing abilities, for example, the Cambridge Face Memory Test, the Benton Facial Recognition Test, Recognition Memory Test for Faces, or the Bielefilder Famous Faces Test (Duchaine, Germine, & Nakayama, 2007; Duchaine & Nakayama, 2006; Kennerknecht et al., 2006) Based on our earlier analysis of congenital amusia, it should not be surprising that with a 2 *SD* cut off and a normal distribution of scores, the reported prevalence of developmental prosopagnosia is around 2%–2.5% (Grüter, Grüter, Bell, & Carbon, 2009; Kennerknecht et al., 2006; Van den Stock, van de Riet, Righart, & de Gelder, 2008; Yardley, McDermott, Pisarki, Duchaine, & Nakayama, 2008). In fact, there is no other possibility. Any larger value is simply an indication of the degree of negative skew in the distribution.

In sum, this brief review highlights that the issue of estimating the prevalence of congenital amusia in the population is not unique, but rather is a more general problem. Prevalence reports for dyslexia, dyscalculia, and developmental prosopagnosia are subject to the same criticism we have raised with respect to amusia; that is, the reported rate of occurrence depends on the specific test, cutoff, and degree of skew in the distribution.

[1]Published MBEA norms can be accessed at www.brams.umontreal.ca/plab/publications/article/57#extras (Peretz et al., 2008)

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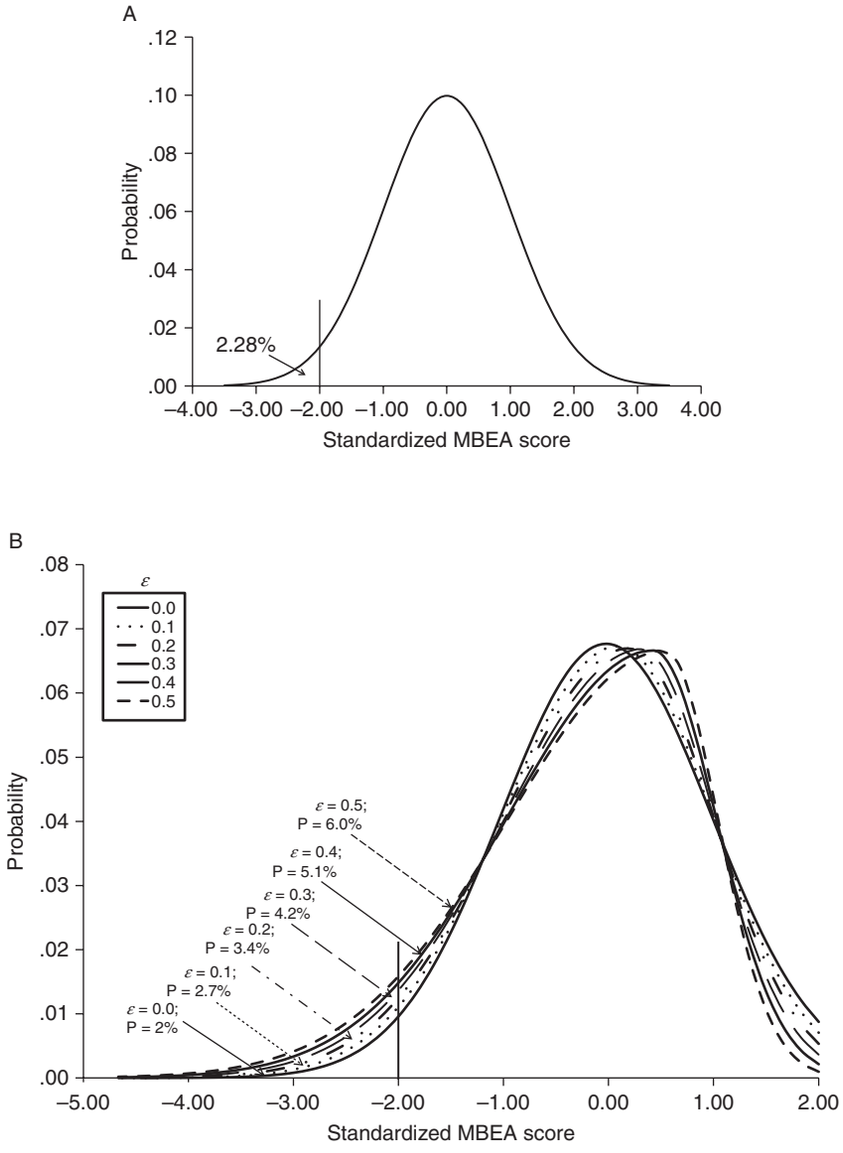

FIGURE 1. (A) A standard normal distribution. Assuming a normal distribution of musical abilities in the population, a 2 *SD* cutoff for the MBEA would necessarily yield an occurrence rate of congenital amusia of 2.28% (Gravetter & Wallnau, 2006). (B) A family of epsilon-skew-normal distributions (Mudholkar & Hutson, 2000). Distributions were generated according to the formula

$$f_0(x) = \begin{cases} \frac{1}{\sqrt{2\pi}} \exp\left(-\frac{(x-\theta)^2}{2(1+\varepsilon)^2}\right) & \text{if } x < 0 \\ \frac{1}{\sqrt{2\pi}} \exp\left(-\frac{(x-\theta)^2}{2(1-\varepsilon)^2}\right) & \text{if } x \geq 0, \end{cases}$$

where θ refers to the mode of the distribution, and ε to the degree of skew. The skew parameter, ε, was varied systematically, and prevalence corresponding to varying amounts of skew were calculated; estimated prevalence (P) for each value of ε is reported in the figure. A fit of the distribution to published norms[1] yielded a value of the skew parameter $\varepsilon = 0.17$ and a prevalence of 3.2%. Estimated prevalence is increased to the reported 4% by modestly adjusting the skew parameter, ε, to 0.26. The epsilon-skew-normal distribution reduces to the standard normal distribution when $\varepsilon = 0$; note that for this distribution, the corresponding percentage value is 2.0% rather than 2.28%. This is due to the truncation of the positive tail of the distribution, as perfect performance on the MBEA corresponds to a *Z* score of +2.0.

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Is there a clear solution to this problem? In short, for test-based methods alone, the answer is no, as estimates of prevalence always will be tied to the specific test used and cutoff. However, one potential approach is the use of model-based methods that rely on theoretically-defined patterns of performance across tests for diagnosis (Akbarzadeh-T & Moshtagh-Khorasani, 2007; Georgopoulos, Malandraki, & Stylios, 2003). A notable strength of these approaches is the use of multiple dependent measures to characterize performance. With respect to congenital amusia, one potential avenue is to more closely consider the separate scores on the various subtests of the MBEA (e.g., to identify individuals showing dissociations between performance on the melodic and rhythmic subtests), rather than relying on a single composite score. It should additionally be noted that diagnosis of, for example, aphasia or prosopagnosia is rarely accomplished without heavy reliance on patient history and structured interviews in addition to test-based assessments. Along these lines, Cuddy and colleagues have begun to expand the study of congenital amusia, administering questionnaires to potentially amusic individuals and using factor analysis to determine the survey items that best predict the diagnosis of amusia (Cuddy et al., 2005). We see recent efforts to combine psychometric assessments of amusia with survey measures as a step in the right direction, but this approach does not represent a panacea for the problem of how to interpret estimates of prevalence.

In conclusion, current test-based methods, such as the DTT and MBEA, used to determine the prevalence of congenital amusia, do not yield estimates that are informative about the true rate of occurrence of these disorders in the general population, but rather yield values that are dependent on the specific test, cutoff, and degree of skew in the distribution of scores. In reaching this conclusion, it is important to add that: (1) we are not diminishing the importance of using tests, such as the MBEA, as a method for assessing musical ability, and (2) we are not describing a problem that is specific to congenital amusia. Indeed, an inspection of the literature shows that the same issue emerges in a range of disorders, including dyslexia, dyscalculia, and developmental prosopagnosia. We do, however, caution against attributing meaning to 4% (or any other absolute percentage) as an estimate of the prevalence of congenital amusia. As our initial brief review indicates, the 4% value, in particular, is becoming so widely cited that, without due prudence, risks becoming an accepted fact.

Author Note

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References

Akbarzadeh-T, M.-R., & Moshtagh-Khorasani, M. (2007). A hierarchical fuzzy rule-based approach to aphasia diagnosis. *Journal of Biomedical Informatics, 40*, 465–475.

Ayotte, J., Peretz, I., & Hyde, K. (2002). Congenital amusia: A group study of adults afflicted with a music-specific disorder. *Brain, 125*, 238–251.

Cuddy, L. L., Balkwill, L.-L., Peretz, I., & Holden, R. (2005). Musical difficulties are rare: A study of "tone deafness" among university students. *Annals of the New York Academy of Sciences, 1060*, 311–324.

Démonet, J., Taylor, M., & Chaix, Y. (2004). Developmental dyslexia. *The Lancet, 363*, 1451–1460.

Douglas, K. M., & Bilkey, D. K. (2007). Amusia is associated with deficits in spatial processing. *Nature Neuroscience, 10*, 915–921.

Drayna, D., Manichaikul, A., de Lange, M., Snieder, H., & Spector, T. (2001). Genetic correlates of musical pitch recognition in humans. *Science, 291*, 1969–1972.

Duchaine, B., Germine, L., & Nakayama, K. (2007). Family resemblance: Ten family members with prosopagnosia and within-class object agnosia. *Cognitive Neuropsychology, 24*, 419–430.

Duchaine, B., & Nakayama, K. (2006). The Cambridge Face Memory Test: Results for neurologically intact individuals and an investigation of its validity using inverted face stimuli and prosopagnosic participants. *Neuropsychologia, 44*, 576–585.

Dykman, R., & Ackerman, P. (1992). Diagnosing dyslexia: IQ regression plus cut-points. *Journal of Learning Disabilities, 25*, 574–574.

Forness, S. R., Sinclair, E., & Guthrie, D. (1983). Learning disability discrepancy formulas: Their use in actual practice. *Learning Disability Quarterly, 6*, 107–114.

Foxton, J. M., Dean, J. L., Gee, R., Peretz, I., & Griffiths, T. D. (2004). Characterization of deficits in pitch perception underlying 'tone deafness'. *Brain, 127*, 801–810.

Foxton, J. M., Nandy, R. K., & Griffiths, T. D. (2006). Rhythm deficits in 'tone deafness.' *Brain and Cognition, 62*, 24–29.

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Georgopoulos, V. C., Malandraki, G. A., & Stylios, C. D. (2003). A fuzzy cognitive map approach to differential diagnosis of specific language impairment. *Artificial Intelligence in Medicine, 29*, 261–278.

Gravetter, F. J., & Wallnau, L. B. (2006). *Statistics for the behavioral sciences* (7th ed.). Belmont, CA: Thomson Wadsworth.

Gross-Tsur, V., Manor, O., & Shalev, R. (1996). Developmental discalculia: Prevalence and demographic features. *Developmental Medicine and Child Neurology, 38*, 25–33.

Grüter, T., Grüter, M., Bell, V., & Carbon, C. (2009). Visual mental imagery in congenital prosopagnosia. *Neuroscience Letters, 453*, 135–140.

Hyde, K., Lerch, J., Zatorre, R., Griffiths, T., Evans, A., & Peretz, I. (2007). Cortical thickness in congenital amusia: When less is better than more. *Journal of Neuroscience, 27*, 13028–13032.

Hyde, K., & Peretz, I. (2004). Brains that are out of tune but in time. *Psychological Science, 15*, 356–360.

Hyde, K., Zatorre, R., Griffiths, T., Lerch, J., & Peretz, I. (2006). Morphometry of the amusic brain: A two-site study. *Brain, 129*, 2562–2570.

Kalmus, H., & Fry, D. B. (1980). On tune deafness (dysmelodia): Frequency, development, genetics and musical background. *Annals of Human Genetics, 43*, 369–382.

Kennerknecht, I., Grueter, T., Welling, B., Wentzek, S., Horst, J., Edwards, S., et al. (2006). First report of prevalence of non-syndromic hereditary prosopagnosia (HPA). *American Journal of Medical Genetics Part A, 140A*, 1617–1622.

Lewis, C., Hitch, G. J., & Walker, P. (1994). The prevalence of specific arithmetic difficulties in 9- to 10-year old boys and girls. *Journal of Child Psychology and Psychiatry, 35*, 283–292.

Lindgren, S. D., de Renzi, E., & Richman, L. C. (1985). Cross-national comparisons of developmental dyslexia in Italy and the United States. *Child Development, 56*, 1404–1417.

Mandell, J., Schulze, K., & Schlaug, G. (2007). Congenital amusia: An auditory-motor feedback disorder? *Restorative Neurology and Neuroscience, 25*, 323–334.

Mudholkar, G. S., & Hutson, A. D. (2000). The epsilon-skew-normal distribution for analyzing near-normal data. *Journal of Statistical Planning and Inference, 83*, 291–309.

Patel, A. D. (2008). *Music, language, and the brain*. New York: Oxford University Press.

Patel, A. D., Foxton, J. M., & Griffiths, T. D. (2005). Musically tone-deaf individuals have difficulty discriminating intonation contours extracted from speech. *Brain and Cognition, 59*, 310–313.

Peretz, I. (2006). The nature of music from a biological perspective. *Cognition, 100*, 1–32.

Peretz, I. (2008). Musical disorders: From behavior to genes. *Current Directions in Psychological Science, 17*, 329–333.

Peretz, I., Brattico, E., Jarvenpaa, M., & Tervaniemi, M. (2009). The amusic brain: In tune, out of key, and unaware. *Brain, 132*, 1277–1286.

Peretz, I., Brattico, E., & Tervaniemi, M. (2005). Abnormal electrical brain responses to pitch in congenital amusia. *Annals of Neurology, 58*, 478–482.

Peretz, I., Champod, A. S., & Hyde, K. L. (2003). Varieties of musical disorders: The Montreal Battery of Evaluation of Amusia. *Annals of the New York Academy of Sciences, 999*, 58–75.

Peretz, I., Gosselin, N., Tillman, B., Cuddy, L. L., Gagnon, B., Trimmer, C. G., et al. (2008). On-line identification of congenital amusia. *Music Perception, 25*, 331–343.

Peretz, I., & Hyde, K. L. (2003). What is specific to music processing? Insights from congenital amusia. *Trends in Cognitive Science, 7*, 362–367.

Ramus, F. (2003). Developmental dyslexia: Specific phonological deficit or general sensorimotor dysfunction? *Current Opinion in Neurobiology, 13*, 212–218.

Särkämö, T., Tervaniemi, M., Soinila, S., Autti, T., Silvennoinen, H., Laine, M., et al. (2009). Cognitive deficits associated with acquired amusia after stroke: A neuropsychological follow-up study. *Neuropsychologia, 47*, 2642–2651.

Shalev, R. S., Auerbach, J., Manor, O., & Gross-Tsur, V. (2000). Developmental dyscalculia: Prevalance and prognosis. *European Child and Adolescent Psychiatry, 9*, 58–64.

Shalev, R. S., & Gross-Tsur, V. (2001). Developmental dyscalculia. *Pediatric Neurology, 24*, 337–342.

Shaywitz, S. E. (1998). Dyslexia. *The New England Journal of Medicine, 338*, 307–312.

Sloboda, J. A., Wise, K. J., & Peretz, I. (2005). Quantifying tone deafness in the general population. *Annals of the New York Academy of Sciences, 1060*, 255–261.

Thompson, W. F. (2007). Exploring variants of amusia: Tone deafness, rhythm impairment, and intonation insensitivity. *Proceedings of the International Conference on Music Communication Science,* 159–163.

Van den Stock, J., van de Riet, W., Righart, R., & de Gelder, B. (2008). Neural correlates of perceiving emotional faces and bodies in developmental prosopagnosia: An event-related fMRI-study. *PLos ONE,* 1–10.

Vicari, S., Finzi, A., Menghini, D., Marotta, L., Baldi, S., & Petrosini, L. (2005). Do children with developmental dyslexia have an implicit learning deficit? *British Medical Journal, 76*, 1392–1397.

Yardley, L., McDermott, L., Pisarki, S., Duchaine, B., & Nakayama, K. (2008). Psychosocial consequences of developmental prosopagnosia: A problem of recognition. *Journal of Psychosomatic Research, 65*, 445–451.

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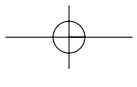

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