An Open Announcement of Corrections: Consolidated Technical, Methodological, and Bibliographic Updates Across Prior Interdisciplinary Preprints (2025–2026)

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*Consolidated Corrigendum & Bibliographic Record: Archival Quality-Assurance Update,
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Abstract

This document issues a formal, consolidated announcement of corrections and archival updates across five previously posted exploratory preprints [1–5] spanning translational autism spectrum disorder (ASD) neurobiology, directed enzyme evolution, circadian–hippocampal modeling, play-based behavioral intervention protocols, and comparative philosophical synthesis. Because those manuscripts were explicitly framed as developing, hypothesis-generating syntheses, the underlying cross-disciplinary research questions remain active lines of inquiry; at the same time, routine archival audit identified technical statements, physiological conventions, study-context descriptions, cultural-protocol elements, and unverified bibliographic entries requiring formal clarification.

Organized manuscript by manuscript, this corrigendum records: (i) assay, probe, and timescale clarifications for molecular and circadian ASD pathways [1, 3]; (ii) biochemical standardization for *Streptomyces lavendulae* BpsA and withdrawal of premature clinical-trial status phrasing [2]; (iii) biomarker polarity alignment and primary-study context updates for play-based developmental protocols [4]; (iv) Indigenous Cultural and Intellectual Property (ICIP) stewardship, deduplication, and personality meta-analysis refinements [5]; and (v) a unified announcement of bibliographic replacements pairing eleven unverified citations with verified peer-reviewed literature. The updated forward-looking ASD research program is presented in our companion open letter [6].

In keeping with transparent scholarly practice, the author issues these consolidated errata, methodological updates, and bibliographic replacements for [1–5].

(1) Corrections to *Perspective: Molecular-Action Pathways for Autism Drug Discovery* [1]. Four technical clarifications apply to [1] alongside six bibliographic updates recorded in Table 1:

- **Gene-target alignment:** *SCN2A*, listed in the abstract alongside *SHANK3* and *SYNGAP1* , is formally incorporated into the main experimental target panel.
- **Organoid maturation stage:** Rather than relying solely on 8–16-week dorsal forebrain organoids (which correspond primarily to mid-fetal neocortex and exhibit metabolic stress [7]), subsequent studies advance to $\geq$ 200–300-day CRISPR-isogenic dorsal–ventral cortical assembloids [8, 9] incorporating medial ganglionic eminence (MGE) interneurons.
- **Pharmacological probe selection:** References to the synthetic REV-ERB agonist `SR9009` are superseded by knockout-controlled clock modulators (e.g., `KL001` and `PF-670462`), reflecting evidence that `SR9009` induces REV-ERB-independent cytotoxicity in double-knockout cells [10].

- **Transcriptomic rhythmicity fraction:** References to >40% circadian rhythmicity denote multi-organ aggregate transcript totals across tissues; within any individual mammalian brain region, $\sim$ 3–10% of transcripts typically display circadian oscillation.

(2) Corrections to *Directed Evolution of the BpsA Carrier Protein Domain* [2]. Three clarifications update the biochemical scope, clinical-status wording, and citation record of [2]:

- **Biochemical classification of BpsA:** Pages 3–5 inadvertently referred to polyketide biosynthesis; throughout [2], *Streptomyces lavendulae* Blue Pigment Synthetase A (BpsA) should be read uniformly (consistent with p. 6 of [2]) as a single-module non-ribosomal peptide synthetase (NRPS) that condenses two L-glutamine molecules into the blue pigment indigoidine. The directed-evolution study adapting BpsA for human PPTase recognition (Brown et al. [11]) was designed as an antibacterial counter-screen rather than a cryptic polyketide or CNS drug-production platform.
- **Clarification of conceptual scenarios vs. human trials:** Discussions of engineered TPH, TH, GAD, BDNF, and neuroligin-1 variants on pp. 23–25 are designated strictly as conceptual and preclinical illustrations. Statements in [2] asserting that human Phase I clinical trials of an evolved TH variant (p. 24) or Phase I/II ASD recruitment for an engineered BDNF variant (pp. 24–25) had commenced are formally withdrawn, and the author notes that [2] reports no new primary mutagenesis kinetics.
- **Bibliographic replacement:** Reference [27] ("Katz et al., 2017") is replaced with Brown et al. (2020) [11] as detailed in Table 1.

(3) Corrections to *Integrating Circadian Rhythm and Neurobiology in Precision Medicine* [3]. Four refinements update the neural-timescale framing, receptor pharmacology, gene-editing strategy, and references of [3]:

- **Separation of circadian and hippocampal timescales:** Hippocampal CA1/CA3 "time cells" [12], which fire over 10^0–10^1 s episodic intervals via theta-paced entorhinal–hippocampal loops, operate on a distinct timescale from 24-hour suprachiasmatic nucleus (SCN) `CLOCK/BMAL1/PER/CRY` transcriptional oscillators. The synchronization phrasing on p. 5 is amended so that circadian–hippocampal interaction is posed strictly as a *cross-timescale modulatory hypothesis* (24-hour CREB/ERK/mTOR phase gating of hippocampal plasticity). Uncited exploratory scenarios regarding optogenetic time-cell rescue (p. 7) and *Syngap1* time-cell coding (p. 10) are explicitly marked as open experimental hypotheses.
- **Melatonergic receptor selectivity:** On p. 2, tasimelteon is corrected from an MT2-selective agonist to a dual MT1/MT2 melatonergic receptor agonist.
- **Gene-delivery vector update:** Because full-length *SHANK3* ($\sim$ 5.2 kb cDNA) and *CHD8* ($\sim$ 7.7 kb cDNA) exceed the $\sim$ 4.7 kb single-AAV packaging ceiling and post-mitotic neurons lack S/G2 homology-directed repair (HDR), the p. 10 Cas9-HDR correction concept is updated to compact CRISPR activation (`dCas9-VP64/p300`) or base/prime editing of the endogenous wild-type allele [13].
- **Bibliographic replacements:** References [8], [9], [11], and [12] of [3] are updated in Table 1.

(4) Corrections to *Play-Based Interventions for Promoting Positive Developmental Outcomes in ASD* [4]. Three updates align the physiological endpoints, literature context, and bibliography of [4]:

- **Autonomic biomarker direction (p. 5):** The phrase "reduced heart rate variability fluctuations" is corrected to **increased heart-rate variability** (elevated RMSSD / HF-HRV), consistent with enhanced parasympathetic/vagal regulation.

- **Primary-study contexts and publication years:** On pp. 2 and 5–6, four study descriptions and dates are clarified: (a) **Lillard et al. (2013)** (Ref. [11] [14]) is clarified as a critical review concluding that causal evidence uniquely linking pretend play to cognitive/social development remains weak under masked controls relative to equifinality or epiphenomenalism; (b) **Gordon et al.** (Ref. [6] [15]) was published in **2013** (not 2011) and tested exogenous intranasal oxytocin during pediatric ASD fMRI rather than a play or cortisol intervention; (c) **Koch et al.** (Ref. [8] [16]) was published in **2014** (not 2015) and is a general meta-analysis of dance movement therapy rather than ASD play cortisol or sleep monitoring; and (d) **van Praag et al. (1999)** [17], cited on p. 6, investigated adult mouse voluntary wheel running rather than pediatric play and is formally added to the bibliography alongside omitted in-text references (Vygotsky 1978; Trevarthen 2001; Steptoe et al. 2008; Seligman 2011; Tomasello 2014).
- **Trial design alignment:** Future clinical deployment of [4] should align with masked-assessor Naturalistic Developmental Behavioral Intervention (NDBI) RCTs (e.g., JASPER, ESDM, Project AIM [18]), pediatric weighted-blanket safety guidance, and autistic participatory co-design tracking masking burden.

(5) Editorial, bibliographic, and cultural-protocol updates to *The Salmon's Revolt Against a Mechanical World* [5]. Four archival adjustments are issued for [5]:

- **Indigenous Cultural and Intellectual Property (ICIP) stewardship:** Out of respect for Worrora, Ngarinyin, and Wunambal custodial protocols in the Kimberley region, Figure 4 (non-custodial contemporary artistic impression of Wandjina figures) is designated for removal from future revisions of [5].
- **Editorial deduplication:** The Eaglehawk and Little Owl fable appearing in both Sections 3 and 5 is consolidated to a single occurrence.
- **Birth-order literature nuance:** Discussion of birth-order personality findings is aligned with Ashton and Lee (2025, *PNAS*, $N > 700{,}000$) [19], who—alongside Rohrer et al. (2015)—reported modest but replicable HEXACO Honesty–Humility and Agreeableness advantages among middle-borns.
- **Reference completion:** Complete author and publication metadata are supplied for Ref. [43] (Stanford Encyclopedia of Philosophy entry on Daoism) and Ref. [45] (Sheeran, Listrom, and Gollwitzer [20]), alongside primary source attributions for William James (1898) and the Advaita *Dashamastvamasi* parable.

(6) Consolidated announcement of bibliographic corrections. Table 1 records the replacement of eleven unverified or conflated entries in [1–3] with verified peer-reviewed citations.

Table 1. Formal announcement of bibliographic replacements across preprints [1–3].

Work & Original Entry	Archival Status	Verified Replacement Citation
[1] Chowdhury & Emmett (2017), *Annu. Rev. Nutr.* 37:135–154	No matching record; pp. 131–156 is Grosso et al. (2017)	J. Bass & M. A. Lazar, *Science* 354, 994–999 (2016); B. Marcheva et al., *Nature* 466, 627–631 (2010)
[1] Wang, Guo et al. (2016), *Mol. Psychiatry* 21:648–656	No matching record; pp. 642–649 is Baumeister et al. (2016)	J. Peça et al., *Nature* 472, 437–442 (2011); P. Monteiro & G. Feng, *Nat. Rev. Neurosci.* 18, 147–157 (2017)
[1] Saad, Lynch et al. (2019), *Neurosci. Lett.* 709:134380	Article 134380 is Hou et al. (2019) on Parkinson's	K. D. Winden, D. Ebrahimi-Fakhari & M. Sahin, *Annu. Rev. Neurosci.* 41, 1–23 (2018)
[1] Tan, Koh et al. (2018), *ACS Chem. Biol.* 13(12):3415–3426	No matching article in Vol. 13(12)	T. Passioura & H. Suga, *Chem. Commun.* 53, 1931–1940 (2017); C. Sohrabi et al., *Nat. Rev. Chem.* 4, 90–101 (2020)

Work & Original Entry	Archival Status	Verified Replacement Citation
[1] Huang, Ramsey et al. (2012), *Immunity* 36(2):196–207	Journal/year mismatch (pp. 188–200 is Harada et al.)	W. Huang, K. M. Ramsey, B. Marcheva & J. Bass, *J. Clin. Invest.* 121(6), 2133–2141 (2011)
[1] Barger, Stefanacci & Semendeferi (2013), *NeuroImage* 75:264–272	Journal/year/topic mismatch	N. Barger, L. Stefanacci & K. Semendeferi, *Am. J. Phys. Anthropol.* 134(3), 392–403 (2007)
[2] Ref. [27] Katz et al. (2017), *Biotechnol. Bioeng.* 114(11):2636–2646	Pages 2628–2636 are Ehrenworth et al. (2017)	A. S. Brown, J. A. Sissons, J. G. Owen & D. F. Ackerley, *ACS Infect. Dis.* 6(11), 2879–2886 (2020)
[3] Ref. [8] Beyens et al. (2017), *Nat. Genet.* 49:511–520	Pages 511–514 are Marsh et al. (2017) on *DCC*	F. F. Hamdan et al., *N. Engl. J. Med.* 360(6), 599–605 (2009); C. Mignot et al., *J. Med. Genet.* 53(8), 511–522 (2016)
[3] Ref. [9] Krystal et al. (2012), *J. Biol. Rhythms* 27(6):433–442	Pages 433–442 are Menegazzi et al. (2012)	Q.-J. Meng et al., *Proc. Natl. Acad. Sci. U.S.A.* 107(34), 15240–15245 (2010)
[3] Ref. [11] Han et al. (2017), *Cell Rep.* 19(4):681–694	Pages 671–679 are Acharya et al. (2017)	R. J. Platt et al., *Cell Rep.* 19(2), 335–350 (2017); A. L. Gompers et al., *Nat. Neurosci.* 20(8), 1062–1073 (2017)
[3] Ref. [12] Inoue & Tsuchiya (2015), *Pharmacol. Ther.* 148:204–216	Vol. 148 ends at p. 198; no matching article	M. D. Ruben, D. F. Smith, G. A. FitzGerald & J. B. Hogenesch, *Science* 365(6453), 547–549 (2019)

(7) Concluding note. Readers citing [1–5] are requested to consult this announcement alongside the constructive translational synthesis in our companion open letter [6].

References

[1] P. De Ceuster, *Perspective: Molecular-Action Pathways for Autism Drug Discovery: Organoids, Circadian Dynamics, and Genetic Engineering*, Zenodo (2025), https://doi.org/10.5281/zenodo.16787215.

[2] P. De Ceuster, *Directed Evolution of the BpsA Carrier Protein Domain for Enhanced Activation by Non-Cognate 4'-Phosphopantetheinyl Transferases Implications for ASD Drug Discovery*, Zenodo (2025), https://doi.org/10.5281/zenodo.17074086.

[3] P. De Ceuster, *Integrating Circadian Rhythm and Neurobiology in Precision Medicine: A Novel Approach for Treating Severe Autism Through the Exploration of Time Cells and Genetic Mechanisms*, Zenodo (2025), https://doi.org/10.5281/zenodo.17074161.

[4] P. De Ceuster, *Play-Based Interventions for Promoting Positive Developmental Outcomes in Autism Spectrum Disorder: A Comprehensive Therapeutic Protocol Informed by Neuroscience, Developmental Psychology, and Positive Psychology.*, Zenodo (2025), https://doi.org/10.5281/zenodo.17074461.

[5] P. De Ceuster, *The Salmon's Revolt Against a Mechanical World*, Zenodo (2026), https://doi.org/10.5281/zenodo.23932794.

[6] P. De Ceuster, *An Open Letter to the Autism-Neurobiology, Molecular-Therapeutics, and Clinical-Intervention Communities: Translational Pathways in Autism Neuroscience — Human-BBB Delivery Evolution, Cross-Timescale Circadian–Hippocampal Coupling, Mature Cortical Assembloids, and Blinded Neurodiversity-Affirming Play Trials*, Companion Open Letter (2026).

[7] A. Bhaduri *et al.*, Nature 578, 142–148 (2020).

[8] F. Birey *et al.*, Nature 545, 54–59 (2017).

[9] A. Gordon *et al.*, Nature Neurosci. 24, 331–342 (2021).

[10] P. Dierickx *et al.*, Proc. Natl. Acad. Sci. U.S.A. 116, 12147–12152 (2019).

[11] A. S. Brown, J. A. Sissons, J. G. Owen, and D. F. Ackerley, ACS Infect. Dis. 6, 2879–2886 (2020).

[12] C. J. MacDonald, K. Q. Lepage, U. T. Eden, and H. Eichenbaum, Neuron 71, 737–749 (2011).

[13] N. Matharu *et al.*, Science 363, eaau0629 (2019).

[14] A. S. Lillard *et al.*, Psychol. Bull. 139, 1–34 (2013).

[15] I. Gordon *et al.*, Proc. Natl. Acad. Sci. U.S.A. 110, 20953–20958 (2013).

[16] S. Koch, T. Kunz, S. Lykou, and R. Cruz, Arts Psychother. 41, 46–64 (2014).

[17] H. van Praag, G. Kempermann, and F. H. Gage, Nature Neurosci. 2, 266–270 (1999).

[18] M. Sandbank *et al.*, Psychol. Bull. 146, 1–29 (2020); BMJ 383, e076733 (2023).

[19] M. C. Ashton and K. Lee, Proc. Natl. Acad. Sci. U.S.A. 122, e2416709121 (2025).

[20] P. Sheeran, O. Listrom, and P. M. Gollwitzer, Eur. Rev. Soc. Psychol. 36, 162–194 (2025).