

## List of publications - Dr. Christian Mayer

My name is in bold, the names of the postdocs and PhD students I supervised are in italics. A full list of my publications can be found on [Google Scholar](#).

- *A. R. Bright, Y. Kotlyarenko, F. Neuhaus, D. Rodrigues, C. Feng, C. Peters, I. Vitali, E. Dönmez, M. H. Myoga, E. Dvoretzkova, and C. Mayer.* “Temporal control of progenitor competence shapes maturation in GABAergic neuron development in mice”. In: **Nat Neurosci** (2025). DOI: [10.1038/s41593-025-01999-y](https://doi.org/10.1038/s41593-025-01999-y) **I have served as a senior author**

This study uncovers how progenitor maturation competence shapes the differentiation of GABAergic neuron subtypes. We identify NFIB as a regulator of chromatin remodeling in late-born progenitors, showing how temporal dynamics of neurogenesis influence molecular identity and vulnerability.

- *J. Zhou, I. Vitali, S. Roig-Puiggros, A. Javed, I. Cantando, M. Puglisi, P. Bezzi, D. Jabaudon, C. Mayer, and R. Bocchi.* “Dual lineage origins contribute to neocortical astrocyte diversity”. In: **Nat Commun** 16.1 (2025). DOI: [10.1038/s41467-025-61829-4](https://doi.org/10.1038/s41467-025-61829-4)

In this study we used the TrackerSeq lineage tracing platform to demonstrate that astrocytes in the cortex arise from not one, but two distinct progenitor lineages, each giving rise to specialized subtypes with distinct roles.

- *I. Adameyko, T. Bakken, A. Bhaduri, C. Chhatbar, M. G. Filbin, D. Gate, H. Hochgerner, C. N. Kim, J. Krull, G. La Manno, Q. Li, S. Linnarsson, Q. Ma, C. Mayer, V. Menon, P. Nano, M. Prinz, S. Quake, C. A. Walsh, J. Yang, O. A. Bayraktar, O. Gokce, N. Habib, G. Konopka, S. A. Liddelow, and T. J. Nowakowski.* “Applying single-cell and single-nucleus genomics to studies of cellular heterogeneity and cell fate transitions in the nervous system”. In: **Nat Neurosci** 27.12 (2024), pp. 2278–2291. DOI: [10.1038/s41593-024-01827-9](https://doi.org/10.1038/s41593-024-01827-9)

This collaborative consensus paper outlines best-practice guidelines for applying single-cell and lineage-tracing technologies in neuroscience. It provides conceptual and technical foundations for studying cell fate transitions and disrupted developmental trajectories in brain disorders.

- *E. Dvoretzkova, M. C. Ho, V. Kittke, F. Neuhaus, I. Vitali, D. D. Lam, I. Delgado, C. Feng, M. Torres, J. Winkelmann, and C. Mayer.* “Spatial enhancer activation influences inhibitory neuron identity during mouse embryonic development”. In: **Nat Neurosci** (2024). DOI: [10.1038/s41593-024-01611-9](https://doi.org/10.1038/s41593-024-01611-9) **I have served as a senior author**

We established a novel *in vivo* framework combining CRISPR perturbation, lineage tracing, and enhancer activity assays to study GABAergic neuron development. We show that MEIS2, a high-risk gene for syndromic autism, regulates inhibitory neuron identity through differential enhancer binding and lineage-specific transcriptional control. These mechanisms may help explain disrupted inhibitory circuit formation in ASD or ID.

- *L. Del-Valle-Anton, S. Amin, D. Cimino, F. Neuhaus, E. Dvoretzkova, V. Fernández, Y. K. Babal, C. Garcia-Frigola, A. Prieto-Colomina, R. Murcia-Ramón, Y. Nomura, A. Cárdenas, C. Feng, J. A. Moreno-Bravo, M. Götz, C. Mayer, and V. Borrell.* “Multiple parallel cell lineages in the developing mammalian cerebral cortex”. In: **Sci Adv** 10.13 (2024), eadn9998. DOI: [10.1126/sciadv.adn9998](https://doi.org/10.1126/sciadv.adn9998)

This study applies barcoded lineage tracing and single-cell RNA-seq in the ferret cortex—a gyrencephalic species—to uncover conserved parallel progenitor lineages across cortical folds. It illustrates how our TrackerSeq method can be applied to species with expanded germinal zones, providing insights into cortical development and lineage architecture relevant to human brain evolution and malformations.

- *R. C. Bandler and C. Mayer.* “Deciphering inhibitory neuron development: The paths to diversity”. In: **Curr Opin Neurobiol** 79 (2023), p. 102691. DOI: [10.1016/j.conb.2023.102691](https://doi.org/10.1016/j.conb.2023.102691) **I have served as a senior author**

This review synthesizes emerging insights from single-cell genomics on how GABAergic neuron diversity arises during development. It highlights molecular trajectories of subtype specification and how their disruption may impair inhibitory circuit formation.

- *C. Peters, S. He, F. Fermani, H. Lim, W. Ding, C. Mayer, and R. Klein.* “Transcriptomics reveals amygdala neuron regulation by fasting and ghrelin thereby promoting feeding”. In: **Sci Adv** 9.21 (2023), eadf6521. DOI: [10.1126/sciadv.adf6521](https://doi.org/10.1126/sciadv.adf6521)

By combining single-nucleus RNA sequencing with *in vivo* imaging, we identified transcriptomic subtypes of amygdala neurons linked to feeding behavior. This work provides a framework to connect molecular identity with internal state regulation and motivated behaviors.

- *R. C. Bandler, I. Vitali, R. N. Delgado, M. C. Ho, E. Dvoretzkova, J. S. Ibarra Molinas, P. W. Frazel, M.*

- Mohammadkhani, R. Machold, S. Maedler, S. A. Liddel, T. J. Nowakowski, G. Fishell, and **C. Mayer**. “Single-cell delineation of lineage and genetic identity in the mouse brain”. In: **Nature** 601 (2022). DOI: [10.1038/s41586-021-04237-0](https://doi.org/10.1038/s41586-021-04237-0) I have served as a senior author
- In this study, we developed TrackerSeq, a method that links clonal lineage to neuronal identity via single-cell sequencing. We showed that fate decisions are lineage-biased at the progenitor level, and that true developmental relationships cannot be inferred from transcriptomic data alone. Clonal analysis revealed convergence and divergence patterns essential for understanding how neurons affected in disease may share developmental origins.
- N. Hoermann, T. Schilling, A. H. Ali, E. Serbe, **C. Mayer**, A. Borst, and J. Pujol-Marti. “A combinatorial code of transcription factors specifies subtypes of visual motion-sensing neurons in *Drosophila*”. In: **Development** 147 (2020). DOI: [10.1242/dev.186296](https://doi.org/10.1242/dev.186296)
  - **C. Mayer** and G. Fishell. “Developing neurons are innately inclined to learn on the job”. In: **Nature** 560 (2018). DOI: [10.1038/d41586-018-05737-2](https://doi.org/10.1038/d41586-018-05737-2)
  - S. Dominguez, L. Ma, H. Yu, G. Pouchelon, **C. Mayer**, G. D. Spyropoulos, C. Cea, G. Buzsáki, G. Fishell, D. Khodagholy, and J. N. Gelinas. “A transient postnatal quiescent period precedes emergence of mature cortical dynamics”. In: **Elife** 10 (2021). DOI: [10.7554/eLife.69011](https://doi.org/10.7554/eLife.69011)
  - **C. Mayer**, C. Hafemeister, *R. C. Bandler*, R. Machold, R. Batista Brito, X. Jaglin, K. Allaway, A. Butler, G. Fishell, and R. Satija. “Developmental diversification of cortical inhibitory interneurons”. In: **Nature** 555 (2018). DOI: [10.1038/nature25999](https://doi.org/10.1038/nature25999)
- This study pioneered the use of single-cell RNA sequencing across developmental time points in brain development to reveal how major interneuron subtypes emerge. We identified MEF2C as a key factor in parvalbumin-lineage differentiation, offering a mechanistic link to MEF2C-associated neurodevelopmental disorders.
- *R. C. Bandler*, **C. Mayer**, and G. Fishell. “Cortical interneuron specification: the juncture of genes, time and geometry”. In: **Curr Opin Neurobiol** 42 (2017). DOI: [10.1016/j.conb.2016.10.003](https://doi.org/10.1016/j.conb.2016.10.003)
  - **C. Mayer**, *R. C. Bandler*, and G. Fishell. “Lineage Is a Poor Predictor of Interneuron Positioning within the Forebrain”. In: **Neuron** 92 (2016). DOI: [10.1016/j.neuron.2016.09.035](https://doi.org/10.1016/j.neuron.2016.09.035)
  - **C. Mayer**, X. H. Jaglin, L. V. Cobbs, *R. C. Bandler*, C. Streicher, C. L. Cepko, S. Hippenmeyer, and G. Fishell. “Clonally Related Forebrain Interneurons Disperse Broadly across Both Functional Areas and Structural Boundaries”. In: **Neuron** 87 (2015). DOI: [10.1016/j.neuron.2015.07.011](https://doi.org/10.1016/j.neuron.2015.07.011)
- This study marks my first use of DNA barcodes to study neuronal lineage. We combined barcode labeling with laser capture and Sanger sequencing to trace interneuron clones across the brain, revealing that clonally related interneurons disperse widely across regions—underscoring the spatial dynamics and flexibility of interneuron development.
- V. Hellier, O. Brock, M. Candlish, E. Desroziers, M. Aoki, **C. Mayer**, R. Piet, A. Herbison, W. H. Colledge, V. Prévot, U. Boehm, and J. Bakker. “Female sexual behavior in mice is controlled by kisspeptin neurons”. In: **Nat Commun** 9 (2018). DOI: [10.1038/s41467-017-02797-2](https://doi.org/10.1038/s41467-017-02797-2)
  - D. Kumar, M. Candlish, V. Periasamy, N. Avcu, **C. Mayer**, and U. Boehm. “Specialized subpopulations of kisspeptin neurons communicate with GnRH neurons in female mice”. In: **Endocrinology** 156 (2015). DOI: [10.1210/en.2014-1671](https://doi.org/10.1210/en.2014-1671)
  - R. Aziz, M. Beymer, A. L. Negrón, A. Newshan, G. Yu, B. Rosati, D. McKinnon, M. Fukuda, R. Z. Lin, **C. Mayer**, U. Boehm, and M. Acosta-Martinez. “Galanin-like peptide (GALP) neurone-specific phosphoinositide 3-kinase signalling regulates GALP mRNA levels in the hypothalamus of males and luteinising hormone levels in both sexes”. In: **J Neuroendocrinol** 26 (2014). DOI: [10.1111/jne.12163](https://doi.org/10.1111/jne.12163)
  - M. Beymer, A. L. Negrón, G. Yu, S. Wu, **C. Mayer**, R. Z. Lin, U. Boehm, and M. Acosta-Martinez. “Kisspeptin cell-specific PI3K signaling regulates hypothalamic kisspeptin expression and participates in the regulation of female fertility”. In: **Am J Physiol Endocrinol Metab** 307 (2014). DOI: [10.1152/ajpendo.00385.2014](https://doi.org/10.1152/ajpendo.00385.2014)
  - N. Eberhard, **C. Mayer**, R. Santic, R. P. Navio, A. Wagner, H. C. Bauer, G. Sperk, U. Boehm, and B. Kofler. “Distribution of alarin immunoreactivity in the mouse brain”. In: **J Mol Neurosci** 46 (2012). DOI: [10.1007/s12031-011-9546-y](https://doi.org/10.1007/s12031-011-9546-y)
  - S. de Croft, R. Piet, **C. Mayer**, O. Mai, U. Boehm, and A. E. Herbison. “Spontaneous kisspeptin neuron firing

in the adult mouse reveals marked sex and brain region differences but no support for a direct role in negative feedback”. In: **Endocrinology** 153 (2012). DOI: [10.1210/en.2012-1616](https://doi.org/10.1210/en.2012-1616)

- **C. Mayer** and U. Boehm. “Female reproductive maturation in the absence of kisspeptin/GPR54 signaling”. In: **Nat Neurosci** 14 (2011). DOI: [10.1038/nn.2818](https://doi.org/10.1038/nn.2818)

This work investigated hypothalamic circuits controlling puberty onset and showed that estrogen signaling in kisspeptin neurons regulates this transition. It provides insight into neuroendocrine timing mechanisms relevant for disorders such as precocious puberty and polycystic ovary syndrome (PCOS).

- **C. Mayer**, M. Acosta-Martinez, S. L. Dubois, A. Wolfe, S. Radovick, U. Boehm, and J. E. Levine. “Timing and completion of puberty in female mice depend on estrogen receptor alpha-signaling in kisspeptin neurons”. In: **Proc Natl Acad Sci U S A** 107 (2010). DOI: [10.1073/pnas.1012406108](https://doi.org/10.1073/pnas.1012406108)