KEY RESULTS OF THE ROUND TABLE

"NEURODEGENERATION: BREAKTHROUGHS AND NOVEL THERAPIES"

Else Kröner Fresenius Stiftung

— *Rewriting the story of ageing brains through early action and new therapies*

Neurodegenerative diseases are set to double within 30 years, turning cognitive decline into a defining challenge of an ageing world. In Alzheimer's disease, pathological changes can be detected up to two decades before symptoms, and some childhood dementias can be identified at or even before birth. The task now is to make early testing routine, learn from people who resist disease despite high genetic risk, and advance gene-targeted therapies that extend healthy lifespan rather than prolong years lived with disability.

Across conditions such as Alzheimer's, Parkinson's, Huntington's, childhood dementias and amyotrophic lateral sclerosis, researchers are uncovering shared mechanisms ranging from toxic protein build-up to disrupted clearance pathways and chronic inflammation. Diagnostics such as blood-based biomarkers and genomic sequencing are rapidly converging with RNA-based treatments, stem-cell approaches and, in future, gene editing, opening the door to tailored interventions far earlier in life.

THE PANEL CALL TO ACTION:

1 — Find the disease decades earlier and act while the brain is still healthy.
Make low-cost blood tests for Alzheimer's risk and expanded newborn screening for childhood dementias part of routine care as tests are validated, so pathological changes are detected before symptoms appear. Replace "wait and see" with early, evidence-based interventions that can delay or prevent degeneration instead of only treating late-stage decline.

2 — Prevent neurodegeneration by learning from the people who do not get sick.
Build international cohorts that track "escapees" – people with high-risk mutations or strong biomarkers who remain cognitively intact far beyond expectations – alongside twin and family studies across childhood and adult dementias. Use insights from these groups to design precise prevention strategies.

3 — Use reversible gene-switching therapies to de-risk future gene editing.
Prioritise RNA and antisense oligonucleotide approaches that can safely dial down disease-causing proteins over years while closely monitoring cognition, brain imaging and safety markers. Move stepwise towards permanent CRISPR-based editing only when brain-wide delivery is technically feasible and reversible approaches have generated multi-year evidence that the chosen gene target does not cause harm.

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