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From Land Dispossession to Brain Injury: Settler-Colonial Determinants of Metal Exposure and Neurological Health in the Strong Heart Study

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Introduction

• American Indian adults in the Strong Heart Study (SHS) carry a disproportionate burden of toxic metal exposure and cognitive decline [1, 2].
• Nearly 90% of cerebrovascular events found on SHS brain MRI are subclinical - undetected by routine clinical care [2].
• The conventional public health response treats this as a biological or behavioral problem; while this project asks a structural question, instead.
• Research question: Which social determinants explain this exposure and cognitive burden, and how do they interact?
• Thesis: Four historically produced determinants- land dispossession, extraction, water-law regulation, and healthcare underfunding- converge to produce this outcome.
• Framework: settler-colonialism as the single upstream structure generating all four determinants, rather than operating as four separate coincidental risk factors [3].
• This reframes cognitive decline from an unexplained health disparity into a traceable policy outcome.

Goals & Objectives of the Work

• Trace how federal land, extraction, and water policy produced the metal exposure documented in the Strong Heart Study.
• Identify the specific molecular mechanisms linking metal exposure to cognitive and cerebrovascular decline.
• Argue that no single determinant is sufficient alone.
• Extend the AHA's 2020 recognition of metals as a cardiovascular risk factor toward cognitive/neurological outcomes [8].
• Contribute a policy-traceable model for Indigenous cognitive health equity research.

Methods

• Literature synthesis across federal Indian policy history, environmental/mining policy, water law, and toxicological/epidemiological research. No new primary data collected.
• Primary anchor: the Strong Heart Study (AZ, OK, ND/SD; initiated 1989).
• Cross-cohort check: MESA, used to test generalizability [7].
• Framework: settler colonial determinants of health model [3].
• Four determinants examined:
    1. Land dispossession (1887 Dawes Act)
    2. Extractive industry siting
    3. Water-law regulatory exemption (SDWA)
    4. Healthcare underfunding (IHS)
• Mechanism review: oxidative stress, neuroinflammation, and mitochondrial dysfunction pathways for arsenic, cadmium, lead, and manganese (full mechanism citations in accompanying paper).
• Every empirical claim is sourced to peer-reviewed epidemiology, toxicology, law, or history.

References

1. Li et al. (2022). Environ Res, 215(Pt 3), 114101.
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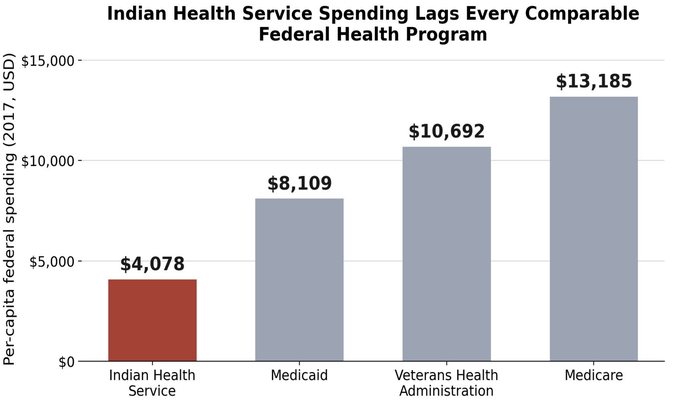
3. Thomas et al. (2025). Public Health Nurs, 42(2), 890–898.
4. National Park Service (2021); Duff-Brown (2025), SIEPR.
5. Hoover et al. (2017). Exposure and Health, 9(2), 113–124.
6. U.S. GAO (2018). Report GAO-19-74R.
7. Domingo-Relloso et al. (2024). JAMA Netw Open, 7(12), e2448286.
8. Breathett et al. (2020). Circulation, 141(25), e948–e959.
Full reference list (24 sources) in the accompanying paper.

Discussion & Results

• Land: the 1887 Dawes Act stripped ~90 million acres (two-thirds of the pre-1887 land base) by 1934; allotment alone raised Native mortality ~20% [4].
• Extraction: >160,000 abandoned hardrock mines sit on Western tribal land; Navajo uranium mining (1944-1986) left >500 abandoned mines.
• Regulation: the Safe Drinking Water Act exempts wells serving <25 people; only the Navajo Nation holds full tribal water-quality primacy.
• Result: 15.1% of unregulated Navajo water sources exceed arsenic standards and 12.8% exceed uranium standards- both worse within 6 km of a mine [5].
• Healthcare: IHS per-capita spending is roughly half of Medicaid's and less than a third of Medicare's [6]- see Figure below.



• Biology: SHS participants show elevated blood cadmium and manganese relative to the general U.S. population [1].
• Cognition: urinary arsenic and cadmium are linked to cerebral atrophy and vascular brain injury in SHS elders [2].
• Generalizability: in the MESA cohort, five urinary metals each independently predict slower processing speed and higher dementia incidence, amplified in APOE4 carriers [7].
• Mechanism: arsenic, cadmium, and lead converge on oxidative stress, neuroinflammation, and mitochondrial dysfunction; manganese accumulates in the basal ganglia and promotes protein misfolding.
• Together, these four determinants connect to the SHS's neurological findings through a documented molecular pathway- not merely a statistical association.

Conclusions & Next Steps

• No single determinant- land, extraction, regulation, or funding- is sufficient alone; each depends on the others to sustain the exposure documented in the Strong Heart Study.
• Genetic modifiers (e.g., APOE4) shape individual severity but do not explain why the population is exposed at elevated levels in the first place.
• Structural history should be treated as a documentable research variable, not background context, in exposure–cognition research.
• Point-of-use interventions (e.g., the Strong Heart Water Study for Private Wells) help but do not substitute for tribal water infrastructure investment or expanded SDWA primacy authority [8].
• Closing the IHS funding gap is necessary to screen for a neurological burden that is already largely subclinical.

Next steps:
• Partner with tribal environmental health programs on community-directed water testing and cognitive-screening initiatives & advocate for expanded tribal SDWA primacy authority and IHS funding parity.
• Contribution: treating environmental justice and Indigenous health equity as inseparable from cognitive health science.

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