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DEMENTIA RISK REDUCTION

TRAUMATIC BRAIN INJURY AND COGNITIVE DECLINE:

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The Public Health Center of Excellence on Dementia Risk Reduction coordinates risk reduction efforts and helps public health agencies share best practices. The Center translates the latest science on dementia risk reduction into actionable tools, materials and messaging that public health agencies can use to reduce dementia risk for all people — including those in diverse, underserved and higher-risk communities.

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WHAT IS ALREADY KNOWN

Each year, there are an estimated 2.5 to 4 million cases of traumatic brain injury (TBI) in the United States. TBI is a disruption in normal brain function caused by a bump, blow, or jolt to the head, or a penetrating injury. These injuries are most common in the youngest and oldest age groups. TBI can raise the risk of dementia in two ways: directly, by making the brain more vulnerable after injury, and indirectly, by increasing the chances of developing other health problems linked to cognitive decline, such as heart disease, epilepsy, and mental health disorders. To lower the risk of TBI and its long-term effects, public health strategies that focus on both preventing injuries and managing chronic health conditions are needed.

The type of injury varies by age. Falls are the leading cause of emergency department visits for children aged 0 to 4 years and for adults aged 65 years and older. For older children and adolescents, being struck by or against an object is the leading cause of TBI, while for older adolescents to middle-aged adults, injuries are related to sports, motor vehicle accidents, or military service.

BACKGROUND AND EVIDENCE BASE

Evidence of an association between TBI and cognitive decline

Acute cognitive effects of TBI typically involve reductions in executive function, processing speed, and learning and memory. The degree to which cognitive recovery in these domains occurs varies, depending on injury severity and other factors (e.g., age, injury characteristics such as diffuse axonal injury or focal intracranial bleed), though recovery is expected in mild TBI (mTBI). Older adults who experience injuries are less likely to fully recover. While cognition can return to preinjury levels after a single TBI, depending on injury severity, risk for dementia may be increased later in life. The associations between TBI and specific dementia subtypes or neuropathology vary, but TBIs occurring in older adults can increase the overall risk of dementia.

The mechanisms underlying the relationship between TBI and later-life cognitive decline are not well understood. TBI is thought to increase risk for cognitive decline later in life through both direct and indirect pathways. These include chronic neuroimmune activation, structural injury that results in cumulative neuronal and cellular dysfunction, and the disruption of the blood-brain barrier as a result of the injury. Polypathology is common at autopsy, particularly in those with moderate to severe TBI, which supports the notion that dementia risk occurs

through multiple processes. Initial studies of biomarkers to identify those at risk for dementia following TBI indicate UCH-L1, ptau181, GFAP, and NfL may have utility in measuring chronic structural brain changes and correlate with cognitive function several years following TBI. However, more research is needed to determine optimal combination and timing of biomarker measurement to assess ongoing chronic brain injury processes.

Genetic vulnerability may also be a key factor. Polymorphism of the APOE gene has received attention in this field, though the evidence is mixed regarding the ε4 allele and long-term outcomes after TBI beyond its recognized contribution to dementia risk. Genome-wide association studies of TBI are limited, though one investigation identified 15 loci, the strongest being NCAM1, APOE, FTO, and FOXP2.

In addition to increased dementia risk due to direct changes associated with TBI, growing evidence suggests that the link between TBI and dementia later in life occurs through indirect pathways as well. Specifically, multiple cross-sectional and longitudinal cohort studies have reported increased rates of chronic conditions that are identified risk factors for dementia, such as cardiovascular/cardiometabolic disease, stroke, seizures, endocrine dysfunction, and psychiatric disorders. Independent of TBI, these comorbid conditions may be risk factors for dementia in the general population. Among those with moderate to severe TBI, greater rates of these comorbid conditions are associated with decline in functional motor and cognitive function over time.

Potential causes of traumatic brain injuries

TBI most commonly results from direct impacts to the head, such as those sustained in motor vehicle accidents, sports, falls, or assaults. These injuries can occur in everyday situations, such as not wearing a helmet while biking or not using a seatbelt in a car. These direct causes are well-recognized and have evidence-based prevention strategies. It is also important to understand social and health-related factors that increase the risk of sustaining a TBI. The following sections explore how adverse childhood experiences (ACEs), violence, and falls contribute to TBI risk, particularly in vulnerable populations.

ACEs: ACEs are potentially traumatic events that a child witnesses or experiences and can include abuse (emotional, physical, or sexual) and household challenges (such as intimate partner violence, substance abuse, mental illness, separation/divorce, and incarceration of a family member). Up to 61% of adults have reported at least 1 type of ACE, and 1 in 6 have reported experiencing four or more types. Women and individuals of racial/ethnic minority groups have a greater risk of experiencing four or more types of ACEs. ACEs can disrupt neurodevelopment and affect health and well-being throughout the lifespan. Health outcomes associated with ACEs, such as homelessness, substance abuse, and violence, can increase a person's risk of experiencing TBI and thereby increase their risk of cognitive decline.

Violence: TBI may result from violence. Abusive head trauma is a leading cause of child abuse deaths in children under the age of five. Domestic, elderly, and intimate partner violence involves unique effects of repetitive or cumulative injury, as well as brain injury effects due to non-fatal strangulation. Survivors of violence are more likely to report ongoing cognitive, physical, and emotional symptoms long-term, and TBI may be underdiagnosed among these survivors because these symptoms can mimic those associated with other mental health or comorbid conditions.

Falls: At least one in four individuals aged 65+ are experiencing falls. Longer recovery among older adults can complicate persistent cognitive sequelae following TBI from cognitive decline associated with pathological aging. Additionally, about 10% to 25% of falls in this population cause fractures, with hip fractures being the most common. Those who experience falls are two to three times more likely to sustain a second fall. Individuals in this age group are likelier to fall indoors than outdoors, and the incidence of falls is even higher among certain populations, such as older adults living in institutions, those recovering from a stroke, and those with diabetes or Parkinson's disease.

IMPLICATIONS FOR PUBLIC HEALTH

Preventing TBI and its long-term effects can be achieved by targeting the circumstances that can precipitate a TBI event, as noted above. Such prevention should be encouraged to preserve brain health in addition to overall general health. Targets must include all ages, relationships and families, and at-risk communities. Social-ecological models and approaches are needed for prevention to be effective in the public health space. Addressing risk factors associated with TBI can have a large and compounding effect on preventing cognitive impairment in older adulthood. These risk factors include falls, violence/abuse, risk taking behaviors, and multifaceted environmental and relational contributions to ACEs. Additionally, continued monitoring and ongoing care directed toward reducing higher rates of chronic conditions associated with dementia risk beyond the post-TBI recovery period is critical.

The role of social determinants of health

The influence of social determinants of health (SDOH) lies at the intersection of poor outcomes following TBI and overall increased risk for dementia. For example, factors such as lower education, lack of health insurance, and lower income are associated with worse cognitive outcomes at one-year post-injury following mTBI. Additionally, living in a disadvantaged neighborhood is linked to more severe and persistent symptoms both shortly after the injury and up to six months later. These social factors have also been identified as independent risk factors for dementia, even among individuals without a history of TBI, after accounting for demographic and background variables. Ultimately, SDOH represents key underlying contributors to the association between TBI, and dementia risk given their position at the intersection of poor TBI outcome following injury and increased dementia risk.

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DISCUSSION

Interventions to reduce the risk of cognitive decline associated with TBI should focus on all age groups, with interventions targeted at the leading causes of TBI by age group. To prevent injuries from accidents in children, playground surfaces should be safe, soft, and composed of appropriate materials. Children should ride in appropriate car seats and booster seats. Adults and children of all ages should wear seatbelts and use helmets and other safety gear for recreational activities. The safety gear should be well maintained, age-appropriate, and worn consistently and correctly. In older adults, multidimensional strategies, such as assistive devices and environmental modifications, should be implemented to prevent and reduce falls. By implementing multi-level public health interventions tailored to different age groups, we can significantly reduce the incidence of TBI and promote better brain health across the lifespan.

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