Post-TBI Nutrition

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Speaker disclosures

✓ Any conflicts of interest?

✓Consulting for Waterson Capitol

The following series planners have no conflicts of interest:

✓ Jennifer Erickson DO

✓ Jess Fann MD

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✓ Chuck Bombardier PhD

✓ Cara Towle MSN RN MA

✓ David Minor

✓ Amanda Kersey PhD

✓ Lauren Miles

Objectives

1. Review pathophysiology of TBI
2. Discuss why diet interventions may be useful
3. Present on available evidence

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Case #1

- 67 yo female Pastor suffered a concussion after tripping over a curb outside of work and hitting her head.
- She experienced several symptoms of concussion that were prolonged in nature, including headache, dizziness, loss of balance, insomnia, and cognitive changes.
- She completed comprehensive neuro rehabilitation with partial improvements in her symptoms.
- She ultimately was unable to keep her job performance where it needed to be, and lost her job (which was associated with her housing).
- After a loss of vocation and home, she took a trip to relax & reset.
- During the trip, she had another concussion. A lower velocity mechanism but nonetheless - triggered her symptoms again.
- She returned to rehab, utilized Parkland pacing for energy conservation, and engaged in sub-symptom threshold aerobic activity.
- About a year out from her first concussion, she decided to change her diet.

Primary & Secondary Injury after TBI

- Primary injury
  - Shock-wave injuries
    - Impact -> shock waves
    - Vibration/resonance of skull after impact -> bruising/contusions
  - Acceleration/deceleration injuries
    - Antero-posterior deceleration (swings cerebrum on the brain stem, stretching/twisting)
    - Rotational acceleration (stretches connections b/w 2 hemispheres)

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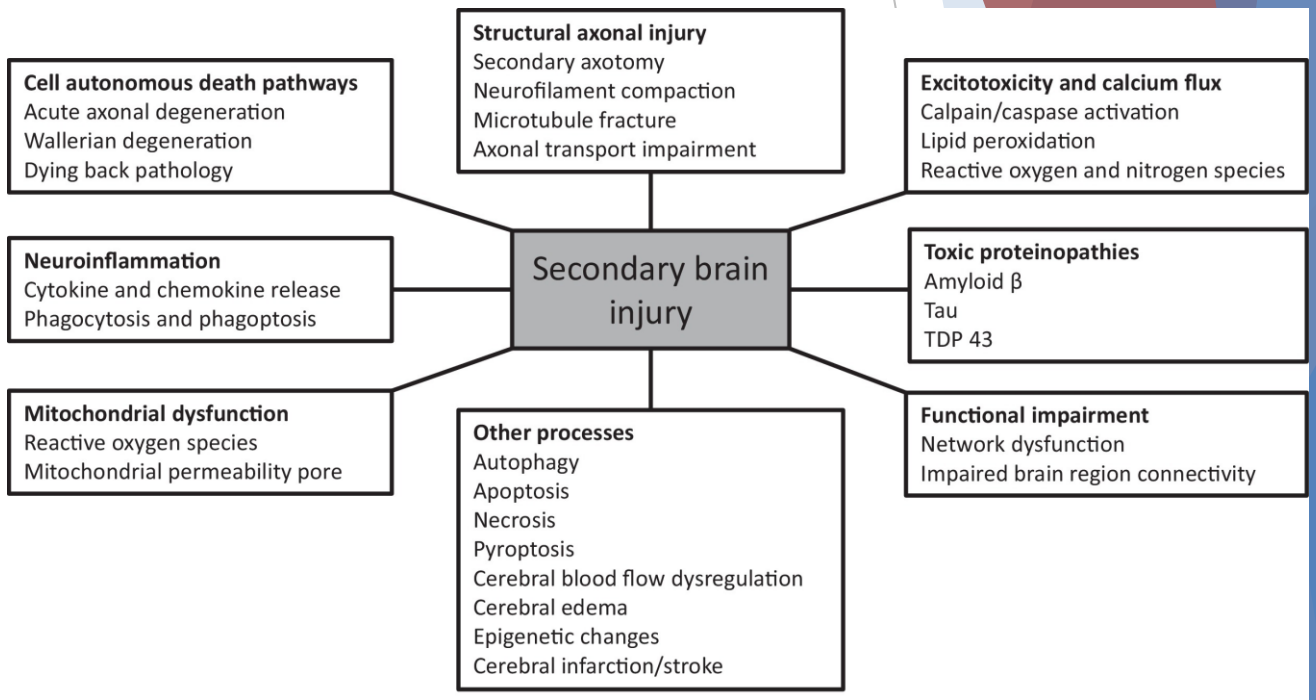
REVIEW ARTICLE

Traumatic brain injury: The first 15 milliseconds

Graham Martin

Accident Compensation Corporation of New Zealand, Wellington, New Zealand

- Secondary injury



Trends in Neurosciences

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Secondary Injury

- Cascade of biochemical, cellular, and molecular events
- Includes ischemia, excitotoxicity, energy failure, cell death. Secondary cerebral swelling. Axonal injury. Inflammation & regeneration.

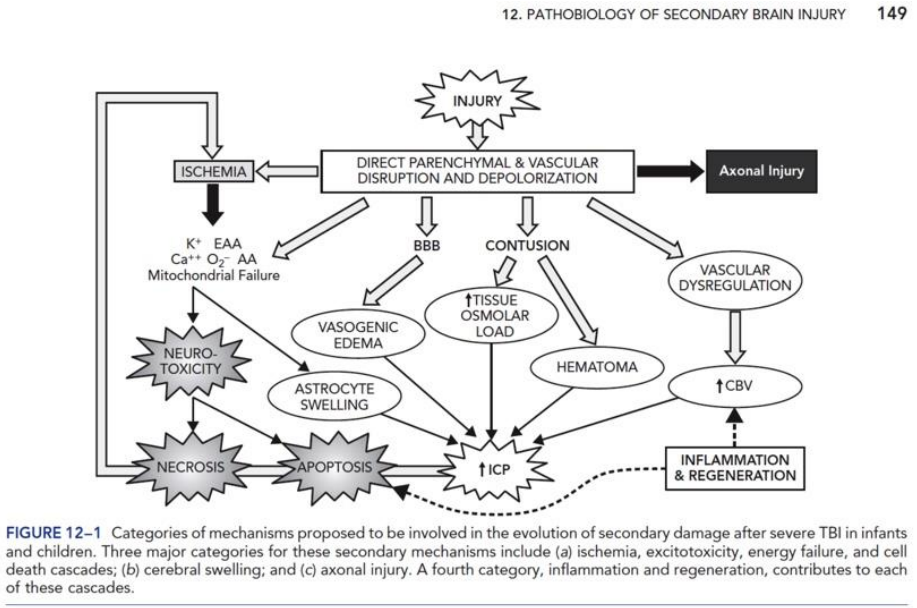
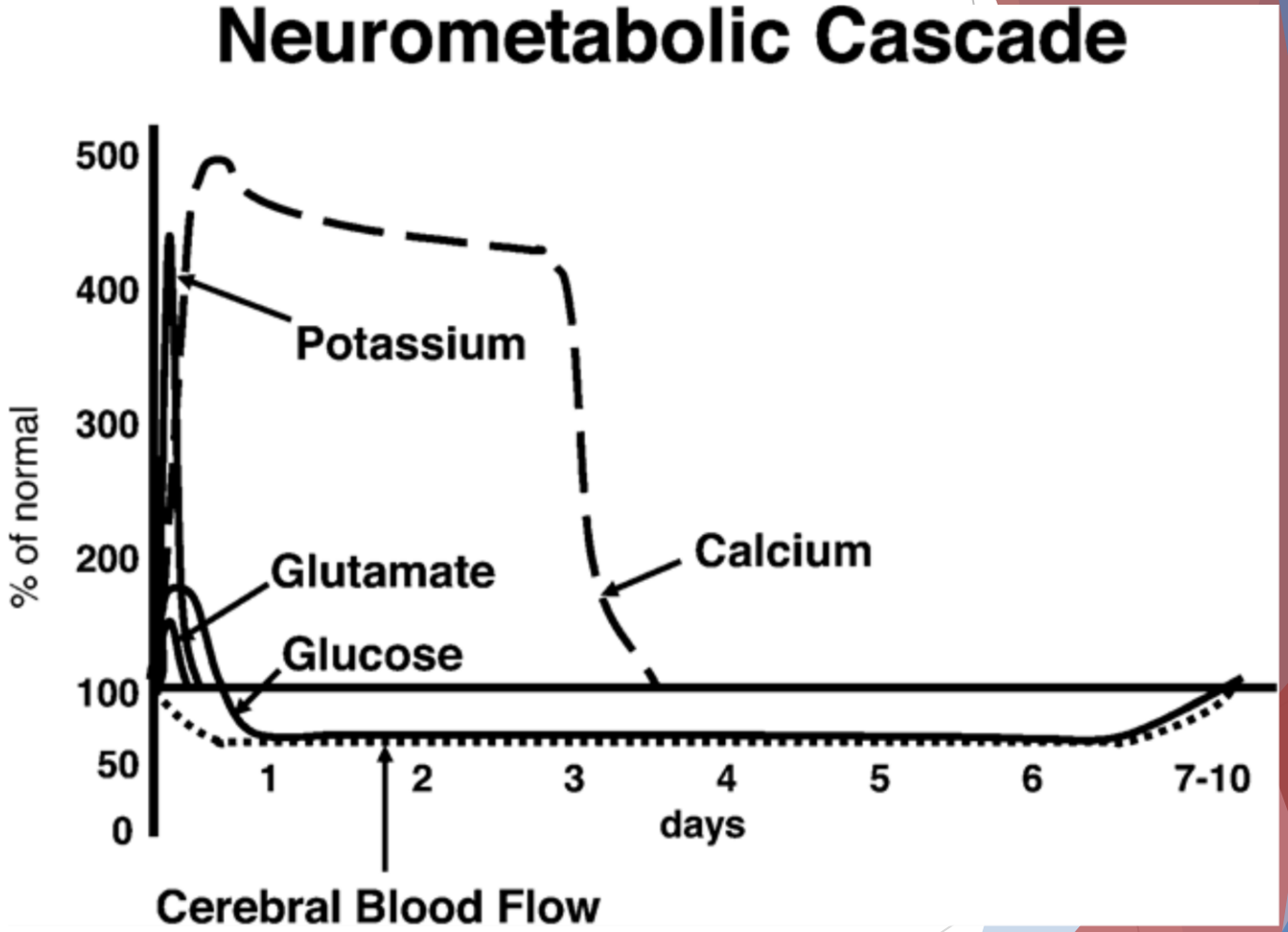


FIGURE 12–1 Categories of mechanisms proposed to be involved in the evolution of secondary damage after severe TBI in infants and children. Three major categories for these secondary mechanisms include (a) ischemia, excitotoxicity, energy failure, and cell death cascades; (b) cerebral swelling; and (c) axonal injury. A fourth category, inflammation and regeneration, contributes to each of these cascades.

Energy failure

- Hyperglycolysis
- Depletion of energy stores
- Reduced energy supply



From: The New Neurometabolic Cascade of Concussion
Neurosurgery. 2014;75(suppl_4):S24-S33. doi:10.1227/NEU.0000000000000505
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So should we use ketones?!

Me in residency

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Research report

The behavioural and pathophysiological effects of the ketogenic diet on mild traumatic brain injury in adolescent rats

Sabrina Salberg [a, b], Himanthri Weerwardhena [a], Reid Collins [a], Raylene A. Reimer [c, d], Richelle Mychasiuk [a, b]

- Glucose becomes “inefficient substrate” following brain injury
- Ketogenic Diet (KD) = high fat, low carbohydrates = ketones replace glucose as main energy source
- Compared KD prior to mTBI and KD after mTBI to sham & standard diet
- Found that pre-KD offered some neuroprotection by reducing balance & motor impairments while increasing exploratory behavior and telomere length.
- Post - KD offered some therapeutic benefit by reducing anxiety & depressive like behaviors

The Effects of a Ketogenic Diet on Behavioral Outcome after Controlled Cortical Impact Injury in the Juvenile and Adult Rat

K. Sofia Appelberg,[1] David A. Hovda,[1,2] and Mayumi L. Prins[1]

- Ketogenic diet x 7 days after a controlled cortical impact injury in both young and old rat
- Therapeutic effect noted in young rat in both cognitive and motor domain
- Age-dependent nature of ketogenic neuroprotection in TBI?
- Postulated to be due to increased ketone production, transporters, and greater enzymatic activity for ketone metabolism in younger brains.

Literature review - 2023
Diet & Mild TBI

- Reviewed pre-clinical & clinical data
- Key points:
  - No strength in evidence to support one type of diet>others.
  - No strength in evidence for regular use of supplements.
  - Animal studies suggest nutrient use can alter recovery patterns.
  - A whole food, nutritious, and high quality diet that supports a healthy body has been shown to help in the recovery of pain, memory, mood and sleep regardless of type of diet. These symptoms are often seen after mTBI.
  - Individuals respond differently to diets. For example, a ketogenic diet can have little impact on lipids in one patient but worsen lipid levels in another.

Homocysteine

- An amino acid that is produced during the breakdown of methionine.
- A natural byproduct of metabolism.
- High homocysteine has been associated with cognitive dysfunction, brain atrophy, dementia, more severe TBI, and lower MOCA scores.
- Levels will become high with:
  - Low levels of vitamin B6, B12, folate, magnesium
  - Kidney or liver dysfunction
  - Medications: methotrexate, azauridine, valproic acid, phenytoin, carbamazepine, sulfasalazine, levodopa, fibrates, bile acid sequestrates (cholestyramine), metformin, OCPs, nitrous oxide, high-doses niacin

Review > Nutrients. 2024 Jul 26;16(15):2430. doi: 10.3390/nu16152430.

Mitigating Traumatic Brain Injury: A Narrative Review of Supplementation and Dietary Protocols

Federica Conti [1], Jackson J McCue [2], Paul DiTuro [3], Andrew J Galpin [4], Thomas R Wood [5] [6]

Affiliations + expand

PMID: 39125311 PMCID: PMC11314487 DOI: 10.3390/nu16152430

- Open access!
- Creatine monohydrate
- Omega-3 fatty acides (DHA & EPA)
- Melatonin

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Mechanisms of action in the brain
Omega 3s

- Decrease neuroinflammation
- Attenuate NFL levels (neurofilament light chain - biomarker for nerve damage & neurodegeneration)
- Preserve brain volume (hippocampus)
- Improve cognitive function
- Possibly neuroprotective preventatively

Mechanisms of action in the brain Creatine monohydrate

- Maintain ATP levels even with high energy demands after TBI
- Support cognition
- Improve symptoms of psychiatric disorders
- Possibly neuroprotective preventatively

Mechanisms of action in the brain BCAAs

- Donates nitrogen in glutamate and GABA production
- Supplementation improves cognition, decreases concussive symptoms, improves sleep disturbances

Mechanisms of action in the brain Choline

- Preserves integrity of BBB & cellular membranes
- Attenuates brain edema
- Precursor to acetylcholine
- Improves spatial & recognition memory performance

Mechanisms of action in the brain Magnesium

- Modulates excitotoxicity
- Regulates intracellular calcium concentrations
- Promotes function and cognitive recovery
- Improves behavioral deficits

Mechanisms of action in the brain Blueberry anthocyanin

- Studied in cog/memory impairment
- Decrease neuroinflammation
- Reduce oxidative stress
- Improve cog function and memory performance
- Regulate BDNF in brain tissue

Vitamin D

- Supplementation can improve cognitive and function outcomes in patients with mild to moderate TBI who were deficient at the time of injury
- Caution supplementing in renal disease & hypercalcemia

Vitamin C

- Serum levels drop after TBI
- Pre-clinical studies: high dose supplementation could be neuroprotective
- But gaps in knowledge for using high dose IV ascorbic acid - not recommended
- Long term high dose supplementation has been associated with increased risk CV mortality in postmenopausal women with diabetes, kidney stones in those with pre-existing hyperoxaluria, and iron overload in those with hereditary hemochromatosis.

Preclinical

- CoQ10
- Curcumin
- Polyphenols (resveratrol, quercetin)
- S-adenosyl methionine (SAMe)
- Vit B2 (riboflavin)
- Vit B3 (niacin)
- Vit B6 (pyridoxine)
- Vit B9 (folic acid)

Nutrition

Diet

Supplements

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Table 2.

Summary of the proposed nutritional and supplementation protocol for the prevention and treatment of TBI.
GI = gastrointestinal; N/A = not applicable.

Nutrient/ Biological Compound	Recommended Intake and Supplementation Strategy	Adverse Effects	Food Sources and Corresponding Amounts Per 100 g
Nutritive compounds derived or available from food			
Omega-3 fatty acids (DHA and EPA)	2–4 g/day of combined DHA and EPA (of which 2 g from DHA)	None	Salmon (2.15 g/100 g cooked) Herring (2 g/100 g cooked) Sardines (1.4/100 g canned) Mackerel (1.2/100 g cooked) Trout (1 g/100 g cooked)
Creatine monohydrate	4 × 5 g/day (20 g/day total)	Potential mild GI distress with doses > 10 g	Beef (600 mg/100 g cooked) Chicken (520 mg/100 g cooked) Herring (1.1 g/100 g cooked) Salmon (600 mg/100 g cooked) Tuna (535 mg/100 g cooked) Cod (400 mg/100 g cooked)
BCAAs	Up to 54 g/day	Potential mild GI distress with supplemental daily doses > 45 g	Meat and poultry (3.6 g/100 g) Dairy products (2.37 g/100 g) Cereals and pasta (1.17 g/100 g)
Riboflavin	400 mg/day	None	Beef liver (3.4 mg/100 g) Fortified cereals (4 mg/100 g)
Choline (as CDP-choline/Citicoline)	1–2 g/day	None	Beef liver (419 mg/100 g) Hard boiled eggs (294 mg/2 eggs) Roasted soybeans (125 mg/100 g) Chicken breast (85 mg/100 g)

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Magnesium (any bioavailable form)	400 mg/day	None	Pumpkin seeds (184 mg/100 g roasted) Chia seeds (131 mg/100 g) Almonds (94 mg/100 g roasted) Spinach (78 mg/100 g boiled)
Blueberry anthocyanins	250–400 mg/day	None	Low-bush wild blueberries (487 mg/100 g)
Non-nutritive compounds			
Boswellia serrata	3 × 400 mg/day	None	N/A
Enzogenol	1 g/day	None	N/A
NAC	4 g/day for 4 days (2 × 2 g), then 3 g/day (2 × 1.5 g).	None	N/A
Melatonin	2 mg at night	None	N/A

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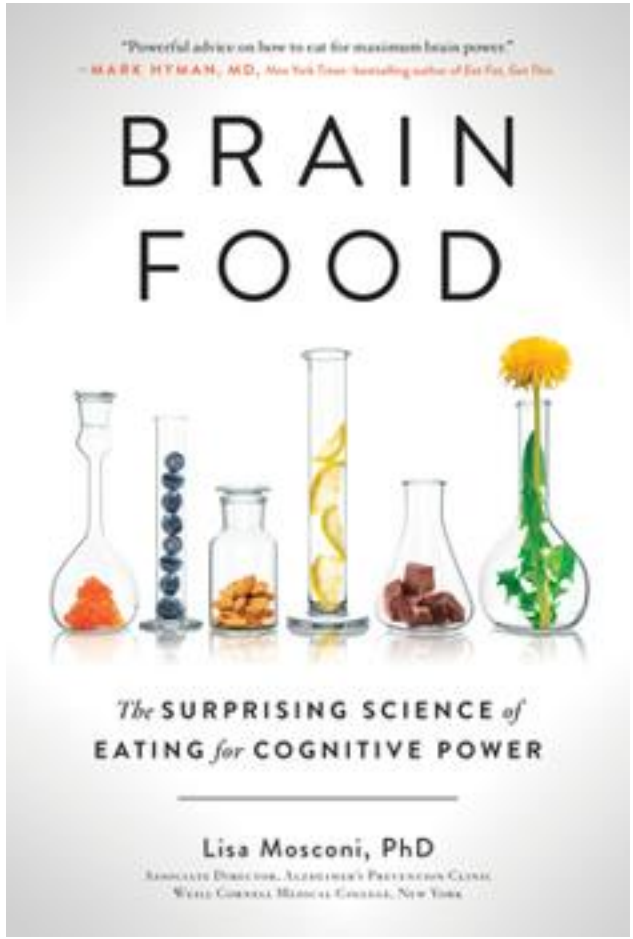
Case # 1



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Thank you!

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