

# Supporting Mental Agility

by Dan Moran, Ph.D.

Though great variability exists, a common feature of aging is a loss of mental agility in learning and memory.



How the brain learns, creates and stores memories is phenomenal; only recently have significant investigative breakthroughs been made on this subject. We all experience a memory but what does a memory look like when stored in the brain? There is much still to learn and explore.

Learning is the ability to process and respond to new information; it requires both short- and long-term memory storage. Of the many regions of the brain involved in memory formation, the role of the hippocampus is to facilitate and integrate memory. It is this region of the brain that, when damaged, can result in defects in memory and the ability to learn.

The network of nerves and nerve cells found in the hippocampus and other areas of the brain undergo physical and chemical changes when we learn. These changes increase the number, size and shape of nerve connections in the brain, as well as the strength of signals that travel across these connections. The degree of permanency of these changes determines whether the memories

made during learning will be long- or short-term. As we age, the number and properties of nerve cells are generally retained in the brain. However, in the hippocampus they may weaken, resulting in impairments in thought and memory.

Since the properties of nerve cells are partly determined by the movement of calcium across the cell membrane, changes in calcium regulation contribute to age-associated declines in learning and memory. Proper management of calcium is known to help establish a memory and activate beneficial changes in both the structure of brain cells and their connections to other cells.

Brain cells use microscopic calcium ions to communicate from one cell to another. An increase in calcium ion channels tends to form at nerve cell connections, permitting more calcium to enter the cell. Poor management of calcium within and outside of the cell could result in harmful effects. The brain uses calcium-binding proteins to manage excess calcium, sopping up calcium

like a sponge. These binding proteins are seriously reduced in the aging brain; therefore, calcium entering the cell is not bound and has a potentially toxic effect. Finally, the pumps involved in either seizing the internal calcium stores or ejecting the excess calcium are compromised in aging brain cells.

Fortunately, calcium levels in the brain can be managed. Using chelator chemicals such as EDTA or EGTA, and naturally occurring calcium-binding proteins such as human calbindin-D28 and apoaequorin (Prevagen) from jellyfish can reduce calcium's deleterious effects, often improving memory and learning tasks. In addition, phosphatidylserine (PS) has been shown to help restore normal memory. Piracetam (a GABA derivative) may improve neuronal activity and decrease important receptor molecules that govern calcium activity. The phytochemical vinpocetin increases blood flow to the brain, effectively increasing the flow of nutrients, clearing the brain of toxins and reducing cell death. And acetyl-L-carnitine increases cell energetics and reduces toxic fatty acid accumulation, improving cell health and survival.

Finally, nothing takes the place of clean air, water and exercise for the health of the brain. Reducing sugar intake, increasing fruits and vegetables in the diet, and moderating negative lifestyle choices such as smoking and alcohol overindulgence add to improved brain health and longer life.

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