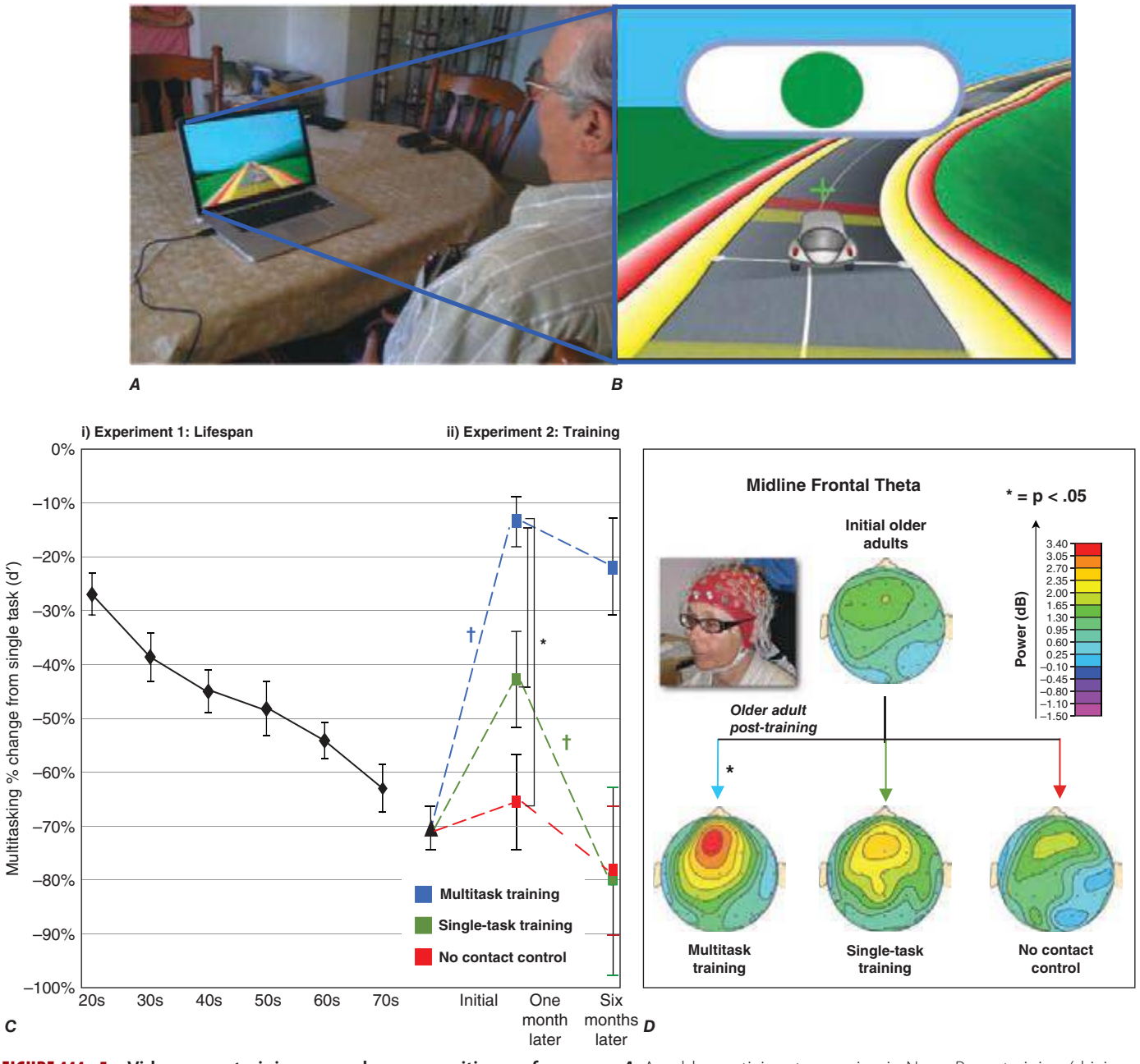

FIGURE 444e-5 **Video game training can enhance cognitive performance.** ***A.*** An older participant engaging in NeuroRacer training (driving while responding to target signs), with (***B***) a screen shot of the experimental training session. ***C.*** NeuroRacer multitasking costs for target discrimination increased (i.e., a larger percentage decrease from single task performance when multitasking) in (*i*) a linear fashion across the lifespan and with (*ii*) costs before training 1 month after training and 6 months after training, showing a differential benefit of multitasking training compared to a no-contact control group and a single-task training group. ***D.*** Midline frontal theta activity obtained with electroencephalogram showed significantly enhanced activity only following multitasking training, mimicking the pattern of change in the behavioral data as well as performance improvements on untrained tests of working memory and sustained attention (not presented). For details, see JA Anguera et al: Nature 501:97, 2013.

Although the number of prions identified in mammals and in fungi continues to expand, the existence of prions in other phylogeny remains undetermined. Some mammalian prions perform vital functions and do not cause disease; such nonpathogenic prions include the cytoplasmic polyadenylation element binding (CPEB) protein, the mitochondrial antiviral-signaling (MAVS) protein, and T cell–restricted intracellular antigen 1 (TIA-1).

All mammalian prion proteins adopt a β-sheet-rich conformation and appear to readily oligomerize as this process becomes self-propagating. Control of the self-propagating state of benign mammalian prions is not well understood but is critical for the well-being of the host. In contrast, pathogenic mammalian prions appear to multiply exponentially, but the mechanisms by which they cause disease are poorly defined. We do not know if prions multiply as monomers or as oligomers; notably, the ionizing radiation target size of PrP^{Sc} prions seems to suggest it is a trimer. The oligomeric states of pathogenic mammalian prions are thought to be the toxic forms, and assembly into larger polymers, such as amyloid fibrils, seems to be a mechanism for minimizing toxicity.

To date, there is no medication that halts or even slows a human neurodegenerative disease. The development of drugs designed to inhibit the conversion of the normal precursor proteins into prions or to enhance the degradation of prions focuses on the initial step in prion accumulation. Although several drugs that cross the blood-brain barrier have been identified that prolong the lives of mice infected with scrapie prions, none have been identified that extend the lives