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Coffee Shops and Fast-food Restaurants: Potential Neighborhood Resources for Cognitive Health and Wellbeing Among Aging Americans

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OBJECTIVES/GOALS: Environmental factors may significantly increase the risk of or buffer against Alzheimer's disease and related dementias, yet strategies to address cognitive decline and impairment to date largely overlook the role of neighborhoods. This mixed-methods study is the first to examine potential links between access to eateries and cognitive function. The goal is to inform place-specific interventions to help aging individuals reduce risk for cognitive impairment through neighborhood community and design. METHODS/STUDY POPULATION: Following an exploratory sequential mixed-methods design, seated and mobile interviews with 125 adults aged 55-92 (mean age 71) living in the Minneapolis (Minnesota) metropolitan area suggest that eateries, including coffee shops and fast-food restaurants, represent popular neighborhood destinations for older adults and sources of wellbeing. To test the hypothesis that these sites, and the benefits they confer, are associated with cognitive welfare, we analyzed data from urban and suburban dwelling participants in the REasons for Geographic And Racial Differences in Stroke (REGARDS) study, a national racially diverse sample of older Americans followed since 2003 (n = 16,404, average age at assessment 72 years). RESULTS/ANTICIPATED RESULTS: Qualitative thematic analysis of how older adults perceived and utilized local eateries include sites of familiarity and comfort; physical and economic accessibility; sociability with friends, family, staff, and customers; and entertainment (e.g., destinations for outings and walks, free newspapers to read). Quantitative results from multilevel linear regression models demonstrate a positive association between density of eateries and cognitive functioning. Taken together, these results complicate our understanding of fast-food settings as possible sites of wellbeing through social interaction and leisure activities. DISCUSSION/SIGNIFICANCE OF IMPACT: The results contribute new evidence towards an emerging ecological model of cognitive health. Understanding whether and how retail food environments can help buffer against cognitive decline among older adults provides novel opportunities to promote wellbeing in later life through community interventions and neighborhood design.

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Collagen Dermal Replacement Scaffold Mechanobiology in Treatment of Difficult-to-Heal Wounds

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OBJECTIVES/GOALS: Difficult-to-heal wounds of the skin are a common and costly medical problem. Dermal replacement strategies have emerged as a solution, but a challenge is identification of optimal scaffold parameters. We present a model for assessment of clinical potential of collagen scaffolds for wound healing. METHODS/STUDY POPULATION: In previous animal experiments, we evaluated dermal replacement scaffolds custom-fabricated from fibril-forming collagen oligomer with controlled fibril density (4, 20, 40mg/cm^3) and spatial gradients in rat excisional wounds. Wound contraction and cellularization were monitored by gross and histological image analysis for comparison with model outcomes. We now parameterize the scaffold parameters for use in the mathematical model of wound healing with nonlinear curve fitting. A preliminary chemo-bio-mechanical finite element model including collagen, cells, and an inflammatory signal was adapted to simulate wound healing results. RESULTS/ANTICIPATED RESULTS: Collagen oligomer microstructure was quantified from scanning electron micrographs. A constitutive law for collagen mechanics was fit to experimental uniaxial tensile tests. We have conducted preliminary three-dimensional finite element model simulations to be validated against experimental wound contraction, recellularization, and collagen remodeling data collected from each experimental group. We show the effects of collagen density and stiffness on wound contraction by altering early wound mechanical properties. We anticipate future work to further improve the model of mechanotransduction, inflammation, and recellularization. DISCUSSION/SIGNIFICANCE OF IMPACT: This work represents the first step towards a computational model of wounds treated with collagen scaffold dermal replacements. In turn, the model will be used to explore cell-scaffold interactions for purposes of prediction and optimization of tissue regeneration outcomes.

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DEEP-PRIMED IL-15 SUPERAGONIST IMPROVES ANTIVIRAL EFFICACY OF HIV-SPECIFIC CD8$^+$ T-CELLS IN HUMANIZED MOUSE MODEL

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OBJECTIVES/GOALS: HIV-specific CD8$^+$ T-cells play a critical role in partially controlling viral replication in infected-individuals, but ultimately fail to eliminate infection. Enhancing these T-cell responses through lymphocyte engineering approaches has the potential as a novel therapy capable of achieving durable control or eradication of infection. METHODS/STUDY POPULATION: IL-15 Superagonist (IL-15SA) potently supports the *in vivo* persistence and antiviral activity of adoptively transferred CD8$^+$ T-cells. The Deep-Priming^{TM} technology platform, developed by Torque, allows for loading of immunomodulators onto the surface of T-cells via electrostatic 'nanogels', which slowly release to deliver sustained autocrine immune stimulation without the harmful effects of systemic exposure. Here, we investigate the impact of IL-15SA Deep-Priming on HIV-specific CD8$^+$ T-cells in a humanized mouse model of HIV infection. Humanized mice were generated by engrafting NOD-*scid*-IL2Rg^{null} mice with memory CD4$^+$ T-cells isolated from an ARV-suppressed HIV+ donor. An autologous HIV-specific Cytotoxic T-Lymphocyte (CTL) clone was isolated, and killing potential confirmed. Four weeks post humanization, mice were infected with HIV and received an infusion of unmodified HIV-Specific CTLs, or IL-15SA Deep-Primed HIV-specific CTLs (CTL-DP). T-cell numbers and plasma viral loads were quantified weekly by flow cytometry and qRT-PCR. RESULTS/ANTICIPATED RESULTS: Mice receiving unmodified CTLs trended toward reduced viral loads compared to the No Treatment condition, while mice receiving CTL-DP saw significant, 2-Log$_{10}$ reductions in VL ($p<0.01$). At 41 days post-infection 100% (5/5) of the No Treatment, 66.7% (4/6) of the CTL treatment, and 16.7% (1/6) of CTL-DP treatment mice had

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