

Autologous Tumor-Derived Immunotherapy: A Novel Approach to Eliminating Cancer Without Chemotherapy or Radiation

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Cancer progression is driven by immune tolerance, insufficient co-stimulation, and impaired danger signaling within the tumor microenvironment, limiting anti-tumor immunity. This study presents TUMVAX, a personalized immunotherapeutic platform integrating endogenous danger signals with optimized antigen presentation. Autologous tumor tissue was cryogenically disrupted and mechanically homogenized to release damage-associated molecular patterns (DAMPs), including DNA, RNA, and cytosolic proteins, mimicking necrotic signals that activate innate immunity. These signals enhance antigen uptake and processing by antigen-presenting cells, enabling efficient presentation via the major histocompatibility complex. The inflammatory microenvironment promotes upregulation of B7 costimulatory molecules, ensuring full T-cell activation through dual-signal priming. Centrifugation at 6000 rpm (10–15 min) reduces low-molecular-weight immunosuppressive cytokines (8–30 kDa). Injections total 0.2 mL (0.1 + 0.1 split). A spatiotemporal dosing strategy was applied, with fractionated injections given at 48-hour intervals; the first dose induces localized inflammation, while the second coincides with peak immune activation, amplifying antigen presentation and co-stimulation. Evaluation using Ehrlich Ascites Carcinoma showed tumor burden reduced to ~30–40% within 7–10 days, while controls showed high mortality by day 11. Sequential dosing produced the fastest responses. Re-exposure triggered immune rejection, confirming durable immunological memory. Histopathology revealed extensive necrosis and dense immune infiltration. TUMVAX represents a shift toward active immune re-education, offering a scalable, low-toxicity, patient-specific therapeutic strategy.

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