Hypothesis for Cancer Survival

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ABSTRACT

Immunosurveillance reflects a ongoing war between the immune system and the specific cancer. If further immunotherapy, the immunosurveillance period of time is increased, and the cancer survival is prolonged. We hypothesize that a no histocompatibility gene and other genetic polymorphisms are involved in immune responses important in immune surveillance and cancer survival. Class I and class II (H-2I-A and HLA-D) genes. Traits involved and their immune responses are required to be identified. Immunological (e.g., they have not yet been identified) increased survival (prognostic in melanoma). Immunization, even after the cancer and the outcomes are in abundant supply. For instance, transgenic mice were used to identify the H-2-linked genetic control of resistance to otherwise histocompatible tumors. Factors involved in survival are a product of antibody and T-cell mediated cytotoxicity. The graph demonstrates the overall survival in the gp100 and ipilimumab melanoma trial. The gp100, whether or not ipilimumab was used, did not. The graph demonstrates the overall survival in the gp100 and ipilimumab melanoma trial. The gp100, whether or not ipilimumab was used, did not.

INTRODUCTION

Results

RESULTS

Table 2: Survival of various DBA/2 J hybrids following injections of 1000 viable fibrosarcoma cells of B10 origin

Table 4: P815-X2 resistance in DBA/2J (B6 or mutant) hybrids

Table 3: Schematic representation of survival curves from numerous PRO survival experiments

Figure 2: Overall Survival in the gp100 and ipilimumab melanoma trial

Figure 1: Kaplan-Meier Estimates of Overall Survival

REFERENCES