ON A HYBRID SCALE MODEL OF DOSE-RESPONSE RELATIONSHIPS UNIVERSALLY APPLIED TO VARIOUS DATA OF IONIZING RADIATION EXPOSURE

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OBJECTIVE To evaluate the low dose risk, this is to develop a widely applied method for dose-response data, using a hybrid scale (HS) model that integrates multiplicative and additive interactions.

METHOD The incidence is given by $I(D) = \exp[\alpha + \beta \text{ hyb}(\tau D)] S(D)$, similar to a generalized L-Q dose-response relationship for radiation-induced cancer in the Annex B of the UNSCEAR 1986 Report. Where D is the dose; α and β are model parameters; τ is the effect modifier which changes from a power function to an exponential dose-response; hyb (τD) is the hybrid function defined as $\tau D + \log(\tau D)$; S(D) is the survival probability of cells having an inactivation constant λ and a feedback parameter ρ for repairing the sublethal damaged cells given by $dS/dD = -\lambda S / (1 + \rho S)$ or $\text{hyb}(\rho S) = \delta - \lambda D$, where $\delta = \text{hyb}(\rho)$ if S = 1 at D = 0. These models are called the Generalized Hybrid Scale (GHS) model for I(D), the Hybrid Scale (HS) models for S(D) and F(D), respectively.

The hybrid scale consists of a log scale, a linear scale and their intermediate (purely hybridized) scale and has a positive parameter e.g., ρ or τ. The concept of hybrid scale is important for identifying the effective range of risk control for defense biological systems and radiation protection.

RESULTS Based on the data of Elkind and Sutton (1960), the HS model of S(D) confirmed the good fitting and interpretation of repair ability according to the split time. The HS model of F(D) has been demonstrated to confirm the good fit and consistency among various data including transformations per surviving cell (Borek, 1984). Based on data of reciprocal translocations per cell (0 – 1,200 r) by Preston and Brewen (1973), the GHS model resulted in good fitting, including decomposed S(D) and F(D) from I (D). The GHS model fits very well to the data: Myeloid leukemia incidence of mice (Mole, 1984; Majo et al, 1986); LSS leukemia mortality (Shimizu et al. 1990), LSS solid cancer mortality (Ozasa et al., 2012) and incidence (Preston et al. 2007; Grant et al., 2017), others. The GHS model provided a consistent estimate of the parameters between males and females in the data reported in detail by Grant et al.

CONCLUSION The HS model of S(D) was confirmed to be valid by data of Elkind and Sutton (1960). The HS model of F(D) and GHS model was confirmed to be useful by various data (Preston and Brewen 1973; LSS leukemia and solid cancer data).