**Figure S1**: Functional network of the 84 ADT-RS genes, with significant interaction with ADT, revealing high degree of connectivity for some ADT-RS genes like *REST* and *EZH2*, and several pathways potentially involved in ADT resistance mechanism. *SOX2* and *NANOG* are key transcription factors regulating many ADT-RS genes.

**Figure S2**: Associations between PAM50 groups and ADT-RS signature

**Table Legends**

**Table 1**: Clinicopathological characteristics of training and validation cohorts

**Table 2**: Univariable and multivariable Cox regression of the validation set studying the interaction between ADT treatment and ADT-RS

**Table S1**: List of NEPC related genes used for model discovery

**Table S2**: List of ADT-RS genes and their coefficients in both arms in the final model

**Table S3**: Clinicopathological characteristics of the matched validation set comparing treated-ADT patients to no-ADT treated patients

**Table S4**: Survival c-indices in discriminating metatastic risk for three genomic signatures (ADT-RS, Decipher, cell cycle genes) across the three validation datasets. ADT-RS scores were multiplied by a factor of -1 so that higher scores reflect a higher risk of metastasis for comparison with the two prognostic-only models.

**Table S5**: Marginal ADT by genomic biomarker interaction effect