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Stress reactivity, susceptibility to hypertension, and differential expression of genes in hypertensive compared to normotensive patients

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Although half of hypertensive patients have hypertensive parents, known hypertension related human loci identified by genome-wide analysis explain only 3 % of hypertension heredity. Therefore, mainstream transcriptome profiling of hypertensive subjects addresses differentially expressed genes (DEGs) specific to gender, age, and comorbidities in accordance with predictive preventive personalized participatory medicine treating patients according to their symptoms, individual lifestyle, and genetic background. Within this mainstream paradigm, here, we determined whether, among the known hypertension-related DEGs that we could find, there is any genome-wide hypertension theranostic molecular marker applicable to everyone, everywhere, anytime. Therefore, we sequenced the hippocampal transcriptome of tame and aggressive rats, corresponding to low and high stress reactivity, an increase of which raises hypertensive risk; we identified stress-reactivity related rat DEGs and compared them with their known homologous hypertension-related animal DEGs. This yielded significant correlation between stress reactivity-related and hypertension-related fold changes (log2 values) of these DEG homologs. We found principal components, PC1 and PC2, corresponding to a half-difference and half-sum of these log2 values (Figure 1). Using the DEGs of hypertensive versus normotensive patients (as the control), we verified the correlations and principal components. This analysis highlighted downregulation of β-protocadherins and hemoglobin as whole-genome hypertension theranostic molecular markers associated with a wide vascular inner diameter and low blood viscosity respectively.

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