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Short Communication

Epigenetic in Human Fibroblasts are Chronic Stress-Induced Glucocorticoid and Receptor Activation

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INTRODUCTION

The action of glucocorticoid stress hormones, principally adrenaline in humans, which operate *via* the glucocorticoid receptor, is thought to be a mediator of the genomic effects of stress (GR). Human fibroblasts were sustained exposure to physiological stress levels of cortisol and/or a selective GR antagonist in order to examine how chronic stress driven GR activation affects epigenetic and cell states. Cell proliferation, migration, and morphology were observed to change significantly in response to cortisol; these changes were reversed by concurrent GR blocking. Extensive, but genomic context dependent, alterations in DNA methylation and mRNA expression, particularly at genetic variants known to be involved in cell migration and proliferation, accompanied the GR-driven cell phenotypes. These results give light on how crucial cell phenotypes are shaped by functional epigenomic patterns that are driven by chronic stress [1].

ABOUT THE STUDY

Population Doubling Levels (PDL) at each cell passage and automated cell counts were used to track cell growth. While concurrent relacorilant treatment entirely reversed these effects, cortisol increased the rate of proliferation and increased the proliferative potential of IMR-90 cells [2]. Chronic stress driven glucocorticoid receptor activation modulates mRNA levels of genes with functional roles in fibroblast phenotypes chronic environmental stress can have a significant impact on how cells and the body operate, but the cellular and molecular mechanisms behind these effects are incompletely known. In this study, we demonstrate that sustained exposure to physiological stress hormone levels induces significant, GR-mediated alterations in critical fibroblast characteristics, such as cell proliferation, migration, and shape [3]. This information is significant because it shows that the effects of chronic stress driven GR activation on cells can be seen in dermal fibroblast strains and IMR-90 cells from donors with diverse ancestries and sexes [4]. The GR-driven cell morphologies also coincide with functional methylomic and transcriptome alterations [5,6].

CONCLUSION

With us study is the first to demonstrate that physiological stress levels of cortisol are sufficient to promote not only cell proliferation but also cell migration. Previous research has shown that prolonged exposure to either high non-physiological concentrations of cortisol or synthetic (and much more potent) glucocorticoids extends the proliferative potential of fibroblasts. They conclude that the GR is the main source of the observed stress driven cell phenotypes since these effects are further reversed when the specific GR antagonist relacorilant is used in conjunction with the therapy. In order for an injury to heal, fibroblasts must have the capacity to both proliferate and migrate. Therefore, our results imply that stress driven GR activation could improve their ability to mend wounds, an adaptation that may be required for survival in high stress circumstances. A role for this signature in the chronic stress driven epigenetic control of gene transcription is also suggested by the fact that we have identified gene body hyper methylation as a common signature that extends earlier observations of comparable epigenetic patterns at differentially expressed genes. Chronic stress has been linked to a higher risk of a variety of disease types, such as heart disease, cancer, and mental illness. Our research has shown that exposure to physiological stress hormone levels for an extended period of time has a wide range of impacts at the molecular and cellular levels, with potentially significant implications for human health. These results may be expanded to other human cell types in subsequent research, and it will be important to discover how chronic stress affects cell phenotypes at the systems level and how this affects health and illness.

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