

Featured Research Studies

How does long term exposure to base stations and mobile phones affect human hormone profiles?

<http://www.sciencedirect.com/science/article/pii/S0009912011027330>

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Abstract

Objectives

This study is concerned with assessing the role of exposure to radio frequency radiation (RFR) emitted either from mobiles or base stations and its relations with human's hormone profiles.

Design and methods

All volunteers' samples were collected for hormonal analysis.

Results

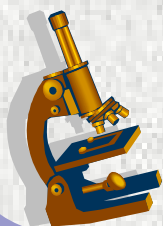
This study showed significant decrease in volunteers' ACTH, cortisol, thyroid hormones, prolactin for young females, and testosterone levels.

Conclusion

The present study revealed that high RFR effects on pituitary–adrenal axis.

Highlights

- ▶ This study is concerned with assessing the role of long-term exposure to high radio frequency radiation emitted either from mobile phones or from base stations and its relations with human's hormone profiles.
- ▶ All volunteers are followed for 6 years and blood samples were collected regularly every 3 years for time intervals of 1 year, 3 years and 6 years for hormonal analysis and the blood samples were taken at 8.0 a.m.
- ▶ This study showed reduction in volunteers' plasma ACTH, serum cortisol levels. Also, they showed decrease in the release of the thyroid hormones especially T3. In addition, each of their serum prolactin in young females (14–22 years), and testosterone levels significantly dropped due to long-term exposure to radio frequency radiation. Conversely, serum prolactin levels for adult females (25–60 years) significantly rose with increasing exposure time.
- ▶ In conclusion, the present study revealed that high radio frequency radiation effects on pituitary–adrenal axis represented in the reduction of ACTH, cortisol, thyroid hormones, prolactin in young females, and testosterone levels.



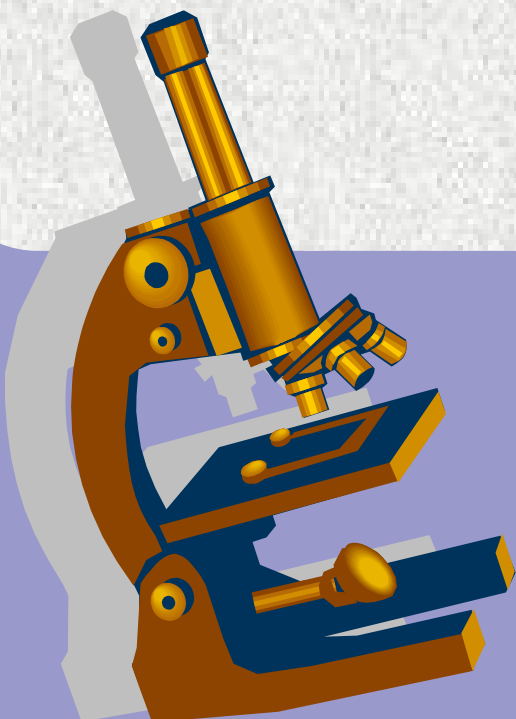
Evaluation of Hepatic Glutathione S-Transferase Mu I and Theta I Activities in Humans and Mice Using Genotype

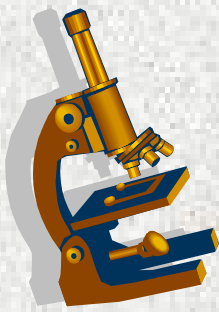
<http://dmd.aspetjournals.org/content/early/2011/12/14/dmd.111.042911.full.pdf+html> (full text)

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Abstract

We investigated the impact of glutathione S-transferases Mu I (GSTM1)- and Theta I (GSTT1)-null genotypes on hepatic GST activities in humans, and compared the results with those of Gstm1- and Gstt1-null mice. In liver with GSTM1/Gstm1-null genotype, GST activity toward p-nitrobenzyl chloride (NBC) was significantly decreased in both humans and mice. Additionally, in liver with GSTT1/Gstt1-null genotype, GST activity toward dichloromethane (DCM) was significantly decreased in both humans and mice. Therefore, null genotypes of GSTM1/Gstm1 and GSTT1/Gstt1 are considered to decrease hepatic GST activities toward NBC and DCM, respectively, in both humans and mice. This observation shows the functional similarity of GSTM1 and GSTT1 toward some substrates between humans and mice. In the case of NBC and DCM, Gst-null mice would be relevant models for humans with GST-null genotype. In addition, decreases in GST activities toward 1,2-dichloro-4-nitrobenzene, trans-4-phenyl-3-buten-2-one, and 1-chloro-2,4,-dinitrobenzene were observed in Gstm1-null mice, and a decrease in GST activity toward 1,2-epoxy-3-(p-nitrophenoxy)propane was observed in Gstt1-null mice. However, an impact of GST-null genotypes on GST activities toward these substrates was not observed in humans. In the case of these mouse-specific substrates, Gst-null mice may be relevant models for humans regardless of GST genotype, since GST activities, which is higher in wild-type mice than in humans, were eliminated in Gst-null mice. This study shows that comparison of hepatic GST activities between humans and mice using genotype information would be valuable in utilization of Gst-null mice as human models.





Impact of GSTM1, GSTT1, GSTP1 polymorphism and environmental lead exposure on oxidative stress biomarkers

<http://www.academicjournals.org/sre/PDF/pdf2011/16Dec/Khansakorn%20et%20al.pdf>
(full text)

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Oxidative stress and genetic related to antioxidants could have influence on susceptibility to lead (Pb) toxicity. In this study, we aim to examine the effects of genetic variations of glutathione S-transferase (GST) gene on oxidative stress alterations (by the measurements of malondialdehyde; MDA and glutathione; GSH) among general population. Real-time-PCR with Taqman probes was performed to analyze GSTM1, GSTT1 and GSTP1 Ile105Val. Blood lead and GSH levels were determined by spectrophotometer. MDA level was measured by HPLC with fluorescence detector. Mean blood Pb level in this study group was 4.85 µg/dl (ranged 2.00 and 18.50 µg/dl). Gender, cigarette smoking and alcohol consumption affected significant blood Pb levels. To further investigation, blood Pb levels were calculated into 3 tertiles and statistical results found only in tertile 3. Individuals with the Val/Val allele for GSTP1-105 had higher blood Pb, and MDA levels but lower GSH level as compared to individuals with Ile/Ile genotype ($p < 0.05$). Similar results were found in GSTM1 deletion, except for the GSH levels. In contrast, no effects of GSTT1 on three parameters were observed. Our findings support consideration of genetic variations of GSH-related genes as the important risk factor for lead toxic effects in the general population with environmental exposure.

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