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Hypogonadism in Males Exposed to Mixed Chemicals in a Mechanic Village in Bodija, Ibadan

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Authors' contributions

This work was carried out in collaboration between all authors. All authors designed the study, participated in the writing of the protocol, read and approved the final manuscript. Authors SUO and SA managed the recruitment of the participants and performed the biophysical measurements, biochemical and statistical analyses. Author SUO wrote the first draft. Authors SUO, MACD and AAO managed the literature, interpreted the data and critically reviewed the manuscript.

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ABSTRACT

Africa has great intensity of chemical exposure and high level of infertility. Functional disorders of the male germ cell and endocrinopathies have been attributed to exposure to mixed chemicals. Data on the mixed chemical exposure on reproductive hormones are sparse in Nigeria. This study was designed to evaluate male reproductive hormones and determinants of occupational exposure to mixed chemicals in a mechanic village in Ibadan.

Forty-three males, auto mechanics aged 18-60 years occupationally exposed to mixed chemicals (MCG) at their work place in Bodija mechanic village with mean±SEM duration of 21.2±1.9 years were age matched with 40 unexposed males (controls) from the University College Hospital, Ibadan and environs. Demography, anthropometry, blood pressure and sexual history were obtained by standard methods. Blood (10 ml) was obtained from each participant for estimation of reproductive hormones by enzyme linked immunosorbent assay while total antioxidant capacity (TAC) was determined spectrophotometrically. Testosterone/oestradiol ratio (TE ratio) was calculated. The MCG were classified based on their reproductive hormone levels into 4 subgroups

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as eugonadic, hypogonadotrophic hypogonadism, sub-optimal hypogonadism and compensated hypogonadism. *P*<0.05 was regarded as significant.
There were significantly raised oestradiol levels, diastolic blood pressure, waist circumference and waist to hip ratio and significantly lower TAC and TE ratio in the MCG than the controls (*P*<0.01). Exposed hypogonadic males exhibited significantly lower LH, FSH, testosterone, prolactin, TAC levels and TE ratio than exposed eugonadic males (*P*<0.05).
Hypogonadism in the males exposed to mixed chemicals may be associated with reduced TE ratio resulting from increased adipose mass as well as oxidative stress associated with the mixed chemical exposure.

Keywords: Mixed chemicals; oxidative stress; hormones; hypogonadism; obesity.

1. INTRODUCTION

Infertility is a growing problem worldwide affecting 8 – 15% of couples in their reproductive age [1]. However, infertility in males is caused by various factors which include environmental toxins, metabolic disorder and testicular blockage [2,3]. Environmental influence such as occupational or accidental exposure to chemicals, use of alcohol, psychotropic drugs, and use of androgenic steroid are capable of exerting profound suppressive effect on the production of sperm and androgens by the testes [4]. Recent evidence suggest that an imbalance between peroxidative and antioxidative substances in serum leads to oxidative stress, resulting in metabolic and functional disorders of male germ cells in some type of infertility [5-7].

Gonadal function is regulated through a feedback mechanism that involves the hypothalamus which sends episodic pulses of gonadotropin releasing hormone (GnRH) to the anterior pituitary gonadotrophic cells which secretes follicle stimulating hormone (FSH) and luteinizing hormone (LH) [8]. LH targets the leydig cells where it stimulates testosterone production and FSH which stimulates spermatogenesis [9]. Abnormal production of prolactin by a pituitary tumour can result in lack of production of LH and FSH by the pituitary with a subsequent drop in testosterone production in the testes [10].

Mixed chemicals are ubiquitous anthropogenic pollutants that are toxic, carcinogenic, and mutagenic to all organisms, including humans [11]. The metabolites of polycyclic aromatic hydrocarbons (PAHs) may bind to proteins and deoxyribonucleic acid (DNA), which causes biochemical disruption and cell damage in animals and cancer in human. The main sources of these contaminants in the environment include forest fire, natural petroleum seeps, combustion of fossil fuels, coal burning, and use of oil for cooking and heating. These environmental contaminants have adverse impacts on male reproductive health [12]. Free radical formation and low antioxidant in occupationally exposed individuals serve as an early biochemical indicator of a pathophysiologic state [13]. Total antioxidant capacity (TAC) measures the antioxidant capacity of all antioxidants in a biological sample, and reflects the antioxidant status of the plasma. Thus, decrease in TAC is a measure of oxidative stress [14,15].

Oxidative stress is a possible mechanism in the toxicity of various chemicals encountered in a work place like mechanic village. Assessment of reproductive hormones and oxidative indices interaction in serum of males occupationally exposed to mixed chemicals is important in predicting those who may develop serious disease including infertility. This study is aimed at evaluating male reproductive hormones and determinants of occupational exposure to mixed chemicals in a mechanic village in Ibadan.

2. STUDY DESIGN

The study was cross sectional conducted in a mechanic village in Bodija, Ibadan. The study protocol was approved by the University of Ibadan and University College Hospital Health Review Committee. Informed consent was obtained from the participants before recruitment.

2.1 Participants

A total of 83 male participants aged 22 to 60 years were recruited for this study. 43 participants were automechanics occupationally exposed to mixed chemicals. The duration of their exposure was mean±SEM duration of 21.2±1.9 years in the mechanic village (MCG) in Bodija, Ibadan. These were age matched with 40 participants who were occupationally unexposed to mixed chemicals and were recruited from

University College Hospital, Ibadan and environs (controls). Demographic and anthropometric factors, sexual history, blood pressure and biochemical characteristics were obtained from each participant. Gonadal factors (Testotosterone, Oestrogen and their ratio (TE ratio) were used as measures of testicular function. FSH, LH and prolactin were measures of pituitary function.

2.2 Demographic Indices, Social Habits, Sexual Potency and Dietary History

Demographic indices, age, social habits (smoking history, alcohol consumption, drugs), sexual potency (libido, nocturnal/early morning erection, other sexual and reproductive history), and dietary history were obtained from a semi-structured pretest questionnaire from each participant.

2.3 Anthropometric and Blood Pressure

Body weight, height, body mass index (BMI) waist circumference (WC), hip circumference (HC), waist hip ratio (WHR), percentage body fat (PBF) and blood pressure (BP) (systolic and diastolic) were obtained from the participants by standard methods as described elsewhere [16,17].

2.4 Biochemical Indices

Blood hormone analysis (FSH, LH, testosterone, prolactin, and oestradiol) was performed in all patients and controls. Hormones were determined using enzyme immunoassay technique (EIA), Immunometrics (UK) Ltd. [18] Total antioxidant capacity (TAC) was determined using ferric reducing antioxidant power (FRAP) assay [19]. TE ratio was calculated.

2.5 MCG Subgroups

Participants were classified based on their reproductive hormone level [2]. The control group was classified as eugonadic (normal LH, FSH and testosterone) based on normal levels of their reproductive hormone. The MCG was classified into four groups. Eugonadic group, hypogonadotrophic hypogonadism (low LH, FSH and testosterone), compensated hypogonadism (high LH, FSH and normal testosterone) and sub-optimal hypogonadism (normal LH, FSH and low testosterone).

2.6 Statistical Analysis

Statistical Package for Social Science (SPSS) 17.0 was used to analyze the data. The mean and standard error of mean (SEM) of normally distributed data was compared using Student's t-test and anova, Chi-square was used to find associations. Data obtained were considered significant at $P<0.05$.

3. RESULTS

There was a significant difference in normal erection during sex in the association of MCG with controls ($P<0.001$) (Table 1).

There were significant differences in intake of vegetable, fruits, dairy products and refined

Table 1. Association of sexual history of males exposed to mixed chemicals with males unexposed to mixed chemicals

Indices	MCG (n = 43)	Controls (n = 40)
Sex desire	Yes = 41(95.3%)[a] No = 2(4.7%)	Yes = 39(97.5%)[a] No = 1(2.5%)
Normal erection during sex	Yes = 28(65.1%)[b] No = 15(24.9%)	Yes = 40(100%)[a] No = 0(0%)
Infertility treatment	Yes = 2 (4.7%)[a] No= 41(95.3%)	Yes= 0(0%)[a] No= 40(100%)
Nocturnal Erection	Yes = 40(93.0%)[a] No = 3(7%)	Yes = 35(87%)[a] No = 5(13%)
Early morning Erection	Yes = 42 (97.7%)[a] No = 1(2.3%)	Yes = 40(100%)[a] No = 0(0%)
Libido	Yes = 38(88.4%)[a] No = 5 (11.6%)	Yes = 37(92.5%)[a] No = 3(7.5%)

a,b superscripts in the same row indicate significant difference at P< 0.05,values are in numbers with percentages in parentheses, MCG = males exposed to mixed chemicals, controls = males unexposed to mixed chemicals

carbohydrate in the association of MCG with controls (P<0.02) (Table 2).

Table 3 shows that MCG had significantly raised DBP, WC and WHR compared with controls (P<0.024)

Table 4 shows that oestradiol was significantly higher while TE and prolactin were lower in MCG than controls (P<0.007). FSH, LH, testosterone and TAC levels were similar in both MCG and controls (P>0.05).

Table 2. Association of dietary history of males exposed to mixed chemicals with males unexposed to mixed chemicals

Indices	MCG (n = 43)	Controls (n = 40)
Vegetables and fruits Intake	Daily= 16(37.2%)[b] Wkly=19(44.2%) Occ. = 8(18.6%)	Daily =19(47.5%)[a] Wkly = 5 (12.5%) Occ. = 16(40%)
Dairy products	Daily = 9(20.9%)[b] Wkly=14(32.6%) Occ. = 10(23.3%)	Daily= 11(27.5%)[a] Wkly= 21(52.5%) Occ. =0(0%)
Refined carbohydrate	Yes=40(93%)[b] No = 3(7%)	Yes=30(75%)[a] No=10 (25%)

a,b superscript in the same row indicate significant difference at p< 0.05, MCG = males exposed to mixed chemicals, controls = males unexposed to mixed chemicals, Wkly = weekly, Occ. = occasionally

Table 3. Comparison of age, blood pressure and anthropometric measures of males exposed to mixed chemicals with males unexposed to mixed chemicals

Indices	MCG	Controls
Age	42.5±1.7[a]	38±1.3[a]
SBP(mmHg)	123.4±1.6[a]	120.9±1.3[a]
DBP(mmHg)	82.4±1.3[b]	78.1±1.0[a]
BMI(kg/m^2)	23.8±0.5[a]	24.1±0.4[a]
PBF (%)	33.0±0.8[a]	32.0±0.8[a]
WC(cm)	86.7±1.7[b]	82.1±1.0[a]
HC (cm)	93.6±1.4[a]	95.1±1.3[a]
WHR	0.9±0.0.1[b]	0.8±0.1[a]
WHtR	19.7±0.4[a]	19.3±0.3[a]

a, b superscript in the same row indicate significant difference p< 0.05, values are in mean±SEM, t=Students's t-test, BMI= body mass index, controls = males unexposed to mixed chemicals, MCG=males exposed to mixed chemicals, PBF= percentage body fat, WC= waist circumference, WHtR= waist height ratio, HC= hip circumference, WHR= waist hip ratio

Table 4. Comparison of reproductive hormones and total antioxidant capacity in males exposed to mixed chemicals with controls

Indices	MCG n = 43 mean±SEM	Controls n = 40 mean±SEM
Testosterone (nmol/L)	13.0±0.6[a]	12.0±0.9[a]
Oestradiol (nmol/L)	1.8±0.2[b]	1.0±0.2[a]
LH (IU/L)	5.1±0.6[a]	6.9±1.5[a]
FSH (IU/L)	5.7±0.8[a]	7.1±0.7[a]
TE ratio	7.2±0.5[a]	12.0±0.7[b]
Prolactin (IU/L)	181±2.3[a]	249±17.9[b]
TAC (µm/L)	1032±70[a]	1186±44.6[a]

a, b superscript in the same row indicate significant difference at p< 0.05,values are in mean±SEM, LH= lutenizing hormone, t = Student's t test, FSH= follicle stimulating hormone, p = probability, MCG = males exposed to mixed chemicals, TE ratio = testosterone oestradiol ratio, TE=testosterone oestradiol ratio, Controls = males unexposed to mixed chemicals, TAC=total antioxidant capacity

Table 5. Total antioxidant capacity, pituitary, sex hormones levels and their ratio in males exposed to mixed chemicals and controls with different gonadal status

Gonadal status	MCG eugonadism n = 30	MCG hypogonadotrophic hypogonadisim n = 6	MCG compensated hypogonadism n = 4	MCG sub-optimal hypogonadism n = 3	Control eugonadism n = 40	Normal reference range
Hormones						
Prolactin	199±20.5	195±66.8	180±42.5	287±123.5	249±17.1	100-390 IU/L
FSH	5.5±0.5	0.7±0.1	15.0±0.4	11.2±3.5	7.1±0.6	2.3- 11.3IU/L
LH	5.0±0.4	0.5±0.2	13.9±2.1	5.3±1.4	6.9±1.4	1.2-12IU/L
Testosterone	14.0±0.6	3.8±0.8	13.9±2.1	3.9±1.2	12.0±0.8	8.42-29 nmol/L
E_2	0.9±0.2	0.6±0.4	1.9±1.1	0.9±0.7	1.0±0.2	0.06-1.5 nmol/L
TE ratio	15.2±0.5	6.3±0.9	7.3±1.0	4.3±1.1	12.0±0.6	
TAC	1066±82.7	664.8±62.7	530±163	610±163.4	1186±42.6	

values are in mean±SEM, LH= lutenizing hormone, FSH= follicle stimulating hormone,T= testosterone, TE ratio = testosterone oestradiol ratio, TAC=total antioxidant capacity, controls = males unexposed to mixed chemicals, MCG = males exposed to mixed chemicals

Table 5 (see above) shows TAC and pituitary, sex hormones levels and their ratio in MCG and control groups with different gonadal status. All males in the control group were eugonadic while varied gonadal status was demonstrated in the MCG group. 13 (30.2%) and 30 (69.8%) of men in the MCG group had hypogonadism and eugonadism respectively. Reduced LH, FSH and testosterone levels indicative of hypogonadotrophic hypogonadism implicating reduced pituitary function were observed in 6 (46.2%) of hypogonadic men. Three (23.1%) had sub-optimal hypogonadism with normal trophic hormones levels but reduced testosterone levels indicative of mild testicular damage or leydig cell failure. Four (30.7%) compensated hypogonadic males in the subgroup exhibited raised trophic hormone levels and normal testosterone suggestive of mild leydig cell failure and early phase of negative feedback removal. The TAC levels of eugonadic, hypogonadotrophic hypogonadic, compensated hypogonadism and sub-optimal hypogonadic MCG groups represent 89.9%, 56.1%, 44.7% and 51.4% respectively of levels observed in controls indicative of oxidative stress.

Table 6 shows comparison of reproductive hormones and TAC levels between eugonadic MCG subgroup and controls (eugonadal).

Table 6. Comparison of reproductive hormones and total antioxidant capacity between eugonadic males exposed to mixed chemicals with eugonadic males unexposed to mixed chemicals

Indices	Eugonadic MCG n = 30 mean±SEM	Controls n = 40 mean±SEM
TAC (µm/L)	1066±82.7[b]	1186.6±44.6[a]
LH (IU/L)	5.0±0.4[a]	6.9±1.5[a]
Testosterone (nmol/L)	13.7±0.6[a]	12.0±0.9[a]
FSH (IU/L)	5.5±0.5[a]	7.1±0.7[a]
Oestradiol (nmol/L)	0.9±0.2[b]	1.0±0.2[a]
Prolactin (IU/L)	199±20.5[b]	249±17.1[a]
TE ratio	15.2±0.5[b]	12.0±0.0[a]

a, b superscript in the same row indicate significant difference p< 0.05,values are in mean±SEM, LH= lutenizing hormone, t = Student's t test, FSH= follicle stimulating hormone, p = probability, MCG = males exposed to mixed chemicals, TE ratio = testosterone oestradiol ratio, TE=testosterone oestradiol ratio, Controls = males unexposed to mixed chemicals, TAC=total antioxidant capacity

TAC, oestradiol and prolactin levels were significantly lower while TE ratio was higher in eugonadal MCG subgroup than eugonadal controls (P<0.03).

Table 7 shows comparison of reproductive hormones and total antioxidant capacity levels between eugonadic and hypogonadotrophic hypogonadism (HH) subgroups of MCG. All reproductive hormones and TAC were significantly lower in the HH subgroup than eugonadal MCG.

Table 7. Comparison of reproductive hormones and total antioxidant capacity levels between eugonadic and hypogonadotrophic hypogonadic subgroup of males exposed to mixed chemicals

Indices	Eugonadic MCG n = 30 mean±SEM	HH MCG n = 6 mean±SEM
TAC (IU/L)	1066±15.16[b]	664.8±26.13[a]
LH(IU/L)	5.0±0.08[b]	0.7±0.08[a][low]
Testosterone (nmol/L)	13.7±0.10[b]	5.6±0.32[a]
FSH(IU/L)	5.5±0.09[b]	1.9±0.05[a][low]
E_2 (nmol/L)	0.9±0.04[a]	0.6±0.15[a]
Prolactin (IU/L)	199±3.77[b]	195±27.83[a]
TE ratio	15.2±0.09[b]	9.3±0.05[a]

a, b superscript in the same row indicate significant difference at p< 0.05, SEM=standard error of mean, values are in mean± SEM, MCG=males exposed to mixed chemicals, FSH = follicle stimulating hormone, LH= lutenizing hormone, T =testosterone, E=oestradiol, TE= testosterone-oestradiol ratio, TAC=total antioxidant capacity, HH=hypogonadotrophic hypogonadism

4. DISCUSSION

Male factor infertility is of global concern currently and has been linked to endocrinopathies associated with exposure to environmental toxic chemicals in the work place. Evidence indicates that exposure to chemicals in the work place generates free radicals which if unaccompanied by appropriate antioxidant status leads to oxidative stress [20]. Environmental influences such as occupational or accidental exposure to chemicals, use of alcohol, psychotropic drugs, and illicit use of androgenic steroids disrupt endocrine milieu and male fertility [4]. In this study, most parameters of sexual potency (early morning erection, libido, and nocturnal erection) were similar between

MCG and controls (*P*>0.05). However, sustained erection during sex in the MCG was significantly decreased (*P*<.001). This might be as a result of hormonal imbalance observed among the MCG. Oestradiol was significantly increased among the mixed chemical group while significant decrease in TE ratio was also observed among the MCG compared with controls (*P*<.007). Oestradiol plays a key role in the development and maintenance of fertility. However, its increased levels are known to inhibit differentiation and proliferation of leydig cells and occupation of testosterone receptor site, block the ability of serum testosterone to induce healthy hormonal signal, increase the production of sex hormone binding globulin that binds to testosterone and decrease fertility potential [21]. Decreased TE ratio in MCG as observed in this study may disrupt spermatogenesis as testosterone is required for spermatogenesis.

Consumption of diets rich in fruits and vegetables enhance total antioxidant capacity and fight against lipid peroxidation [22]. In this study, consumption of vegetables, fruits and dairy products in controls were significantly higher compared with the MCG (*P*< 0.001) while refined carbohydrate consumption in MCG was significantly higher than controls (p< 0.012). High calorie content diet like refined carbohydrate increases BMI and visceral obesity, which are risk factors of cardiovascular disease. Diet rich in antioxidants enhance oxidative status. Antioxidants play a role in decreasing disease risk and maintaining healthy physiologic status [23]. Minimum reactive oxygen species are however necessary to maintain the integrity of the biological system, thus discouraging the use of high antioxidant supplement [24]. Nutritional interventions rich in antioxidants concentrations may be important in imparting the TAC of blood. Consumption of diets rich in fruits and vegetables potentially increase the TAC of healthy individuals as observed in this study.

DBP of the MCG was significantly higher than the controls *(P*<0.01). BMI of the MCG and controls were within normal range and showed no significant difference (*P*>0.01). Central obesity recognized as the main factor involved in pathophysiology of metabolic syndrome (MS) has been postulated to contribute to elevated blood pressure, which is a cardiovascular risk factor [25]. In this study, significantly raised WC and WHR (indicators of visceral obesity) and raised BP were observed in MCG compared with controls indicating enhanced cardiovascular risk in the MCG.

Gonadal status of the MCG showed significant hypogonadism in 13 (30.2%) of men studied. Eugonadal males of MCG exhibited significantly reduced TAC, oestradiol, prolactin but higher TE ratio when compared with controls (*P*<0.03). The reduced TAC levels in the MCG might be as a result of oxidative stress. This is reiterated in the significantly decreased TAC in HH males compared with eugonadic males of MCG. HH males in MCG group (HH MCG) also exhibited significant decrease in LH, FSH, prolactin, testosterone and TE ratio (*P*<0.05). Sex steroids have been shown to have antioxidant role in oxidative stress conditions [26]. Pavlovich et al. reported decreased TE ratio among infertile men with treatable endocrinopathy [27]. Mixed chemicals exposure in mechanic village may have contributed to endocrinopathies and gonadal dysfunction observed in this study.

5. CONCLUSION

Mixed chemical exposure in the auto-mechanics studied might have contributed to the observed endocrinopathies. Hypogonadism in these men may be associated with reduced TE ratio resulting from increased adipose mass as well as oxidative stress, which may be possible mechanisms in the toxicity of these chemicals. Diet rich in antioxidants as well as adequate protective policies in the work place are recommended.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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