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Methamphetamine-associated dysregulation of the hypothalamic–pituitary–thyroid axis

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Abstract

Methamphetamine and HIV impair thyroid function, but few studies have investigated their combined effects on thyroid dysregulation. This study examined the associations of methamphetamine use alone and in combination with HIV on thyroid function among men in South Florida. Measures of thyroid function in methamphetamine-using, HIV-infected (METH+HIV+; n = 127) and HIV-negative (METH+HIV−; n = 46) men who have sex with men (MSM) were compared to non-methamphetamine-using, HIV-negative men (METH−HIV−; n = 136). Thyroid function was dysregulated in methamphetamine-using MSM, irrespective of HIV status. Both meth-using groups had greater odds of abnormal thyroid stimulating hormone levels and significantly higher mean free triiodothyronine (T3) levels. Elevated free T3 was associated with greater depressive symptoms. Overall, outcomes have important implications for assessment of thyroid function in methamphetamine users, particularly among those presenting with depression.

Keywords

Thyroid hormones; HIV; Methamphetamine; Depression; Trauma

Introduction

Methamphetamine (Meth) and Human Immunodeficiency Virus (HIV)-1 infection are co-occurring, intertwined epidemics among men who have sex with men (MSM). MSM who use stimulants have been consistently shown to be at a three to sixfold greater risk of HIV

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Compliance with ethical standards

Conflict of interest Deborah L. Jones, Adam W. Carrico, Suat Babayigit, Violeta J. Rodriguez, Carlos Aguila, and Mahendra Kumar declare that they have no conflict of interest.

Human and animal rights and Informed consent All procedures were in accordance with the ethical standards of the institutional research committees and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. Informed consent was obtained from all individual participants included in the study.

seroconversion (Koblin et al., 2006; Oldenburg et al., 2015; Ostrow et al., 2009; Plankey et al., 2007). Among HIV-infected persons, those who use stimulants such as meth display profound difficulties navigating the HIV care continuum that lead to elevated HIV viral load, faster clinical HIV progression, and greater risk of onward HIV transmission (Carrico, 2011; Carrico et al., 2011, 2014; Ellis et al., 2003; Mayer et al., 2014). There is also recent evidence that meth use is increasing (NDEWS, 2015), and the prevalence of met-h and other stimulant use among MSM in South Florida is among the highest in the United States (Finlayson et al., 2011). Currently, there is no approved pharmacotherapy for stimulant use disorders and behavioral interventions have demonstrated modest effectiveness (Carrico et al., 2016; Colfax et al., 2010). Research addressing important gaps in our understanding of the bio-behavioral pathways whereby meth use is reinforced would inform novel HIV/AIDS prevention approaches.

Negative reinforcement models of addiction propose that increases in depressive symptoms and other forms of negative affect during stimulant withdrawal serve as potent triggers for relapse (Baker et al., 2004). However, relatively little is known about the bio-behavioral pathways whereby meth use may contribute to increases in depressive symptoms. It is also clear that MSM are more likely to have experienced childhood sexual abuse and other traumatic life events that could serve as important risk factors for depression and meth use (O'Cleirigh et al., 2012; Parsons et al., 2012). To date, limited research has focused on whether hypothalamic–pituitary–thyroid axis dysregulation could partially account for meth-associated elevations in depressive symptoms.

Conflicting reports exist regarding the relationship between meth, HIV, and thyroid dysfunction (Noureldeen et al., 2014; Parsa & Bhango, 2013; Passaro et al., 2015), and few investigations have examined this association. Illicit substance use can have adverse effects on thyroid function. For example, meth use disorders can result in autoimmune thyroid diseases (Gozashti et al., 2014). Ingredients in meth such as iodine and lithium can also cause thyroid dysfunction, including hypothyroidism, enlarged thyroids, and hyperparathyroidism. Meth has previously been associated with elevated thyroxine (T4) and serum adrenocorticotrophic hormones as well as lower serum cortisol, triiodothyronine (T3), and thyroid stimulating hormone (TSH) levels (Li et al., 2013). Chronic meth use has also been shown to destroy the regulatory function of the hypothalamic–pituitary–adrenal (HPA) axis (Carrico et al., 2017; Li et al., 2013), which governs normal thyroid function by regulating thyroid hormone secretion, including T3 and T4. Taken together, meth may have direct effect on dysregulation of the hypothalamic–pituitary– thyroid axis.

Impaired thyroid function leads to physiological and emotional dysregulation (Hage & Azar, 2012), and thyroid hormones can be impacted by HIV infection (Hoffmann & Brown, 2007; Mayer et al., 2014). Numerous studies have reported that thyroid dysfunction affects as many as one-third of people living with HIV, which is much higher than in the general population (Ji et al., 2016). Scant research has focused on endocrine dysregulation in meth-using HIV-infected persons, and their combined effects on thyroid dysfunction could have profound physiological as well as psychological consequences. This primary objective of this study was to examine the association of meth use and HIV on thyroid function among MSM in South Florida. It was hypothesized that thyroid function would be most

dysregulated in METH+HIV+ MSM compared with METH+HIV– MSM and METH–HIV– men. We also explored the association of thyroid function with other relevant demographic, psychiatric, and physiological factors.

Methods

Participants and procedures

This study obtained approval from the University of Miami Miller School of Medicine Institutional Review Board and a Federal Certificate of Confidentiality. All procedures were performed in accordance with the ethical standards as laid down in the 1964 Declaration of Helsinki and its later amendments. MSM and male comparison group participants were recruited from clinics, hospitals, support groups, drug treatment programs, and by word of mouth. Data were collected from November 2011 to February 2016 in Miami, Florida.

Participants were eligible for inclusion if they met the following criteria: (a) male and/or MSM (b) between the ages of 18 and 50 years, (c) able to complete the interview in English. Meth users and/or HIV-infected participants were exclusively MSM, due to the increased prevalence of both conditions in this population; control participants were men with any sexual orientation. Exclusion criteria were a history of migraine, seizure, visual impairment, learning disorders, cardiovascular disease, diabetes mellitus, hypertension, bereavement or loss of social support within the last 3 months, current treatment for hepatitis C or major depressive disorder. Written informed consent was obtained from all participants included in the study.

Participants completed a one-time study visit that included the Structured Clinical Interview for DSM-IV Research Version (SCID-I/P) (First et al., 2002), administered by bachelor or master level personnel trained by a licensed psychologist. During the study visit, staff administered a battery of psychological assessments and participants provided a fasting peripheral venous blood sample collected between 9 and 10 in the morning to assess blood glucose levels. All participants were compensated \$50 for their time and transportation.

Participants (N = 347) were assessed; of these, n = 309 were included in the current analysis. Those excluded from the analysis (38; 11%) were due to blood glucose levels greater than 110 mg/dl indicating that they were not fasting samples or were pre-diabetic. For the analysis, the final sample, (N = 309) was divided into three groups (1) meth-using, HIV-infected MSM (METH+HIV+; n = 127); (2) meth-using HIV-negative MSM (METH+HIV–; n = 46); and (3) non-meth-using, HIV-negative men (METH–HIV–; n = 136).

Measures

Demographics and health status—Participants completed a demographic questionnaire assessing age, race/ethnicity, and health-related factors. All participants underwent a physical examination which included weight and height, which were used to calculate body mass index (BMI).

Free T3, free T4, and TSH—Participants provided a fasting blood sample to assess thyroid hormones. Plasma free T3, free T4, and TSH were assessed by ELISA; blood

samples were collected in EDTA tubes that were centrifuged at 3500 rpm for 15 min at 4 °C. The collected plasma was then distributed in a micro centrifuge tube and stored at – 20 °C until assayed. Abnormal levels of free T3, total T3, total T4 and TSH were established using cutoffs from previous research. Normal values for free T3 were defined as 2–4.2 pg/mL; free T4, 7.1–18.5 pg/mL; TSH, 0.3–3.8 mIU/L (Pingitore et al., 2005); all other values were considered abnormal.

Psychiatric factors—Depression was assessed using the 20-item Center for Epidemiologic Studies of Depression (CES-D) self-report measure (Radloff, 1977). Childhood trauma was assessed using a modified version of the Childhood Trauma Questionnaire, specifically the physical and sexual abuse sub-scales; a cutoff of > 13 was used for these subscales, representing severe to extreme childhood trauma (Bernstein et al., 1997). Substance abuse or dependence was assessed using the SCID-I/P (First et al., 2002).

Analytic approach

Analyses of variance (ANOVA) and Chi square tests were utilized to examine Meth/HIV group differences. Post hoc analysis of a significant ANOVA *F* test was conducted with the Bonferroni correction to reduce the family-wise error rate. Bivariate correlations were used to measure the strength of linear relationship between variables. Logistic regression models were conducted to examine correlates of abnormal free T3, free T4, and TSH. The strength of the association assessed by the logistic regression was provided by the adjusted (AOR) with 95% confidence interval (CI). Mediation models were tested including the indirect effect of HIV+METH+ and HIV–METH+ on depression through free T3 using SPSS 24 (statistical software for Windows) to perform all analyses. Using Mplus 7.4, we tested for mediation by estimating the indirect effects of Meth/HIV group via free T3 on higher depressive symptoms. A cutoff value of $p < 0.05$ was used to determine statistical significance.

Results

The mean age of participants was 36.8 (SD = 9.9); slightly more than half of participants (51%) were Hispanic; 26% were black and 19% Caucasian; others identified as biracial, multiracial, or other. Approximately 50% reported an income less than US \$500. Approximately 39% of participants were overweight and 23%, were obese. The Meth/HIV groups differed by age, race, monthly income, body mass index, free T3, childhood abuse, and depressive symptoms. Although there were no Meth/HIV group differences in mean TSH, the prevalence of clinically elevated TSH (i.e., hyperthyroid) was 2.75-fold greater among meth-using MSM versus METH–HIV– men (12.1 vs 4.4%). Similarly, six (3.5%) meth-using MSM displayed clinically low TSH (i.e., hypothyroid) compared to zero (0%) METH–HIV– men. Further demographic detail and comparisons by meth/HIV group status are presented in Table 1.

Using binominal logistic regression, a history of childhood abuse (adjusted odds ratio [AOR] = 0.97 [0.95, 0.99]) was associated with lower odds of abnormal free T3. Older age (AOR = 1.05 [1.02, 1.09]) and being African American (AOR = 2.85 [1.26, 6.42]) were associated

with greater odds of abnormal free T4. Being African American (AOR = 0.26 [0.09, 0.78]) or Hispanic/Latino (AOR = 0.20 [0.08, 0.52]), and having a polysubstance use disorder (AOR = 0.38 [0.15, 0.95]) were associated with lower odds of abnormal TSH values, whereas METH+HIV+ group membership (AOR = 4.51 [1.34, 15.24]) and METH+HIV– group membership (AOR = 8.87 [2.46, 32.00]) were independently associated with greater odds of abnormal TSH values. Details of comparisons are presented in Table 2.

Finally, we tested whether thyroid dysfunction mediated the association of Meth/HIV group with greater depressive symptoms. Greater free T3 ($r = 0.18$, $p < .01$) and lower free T4 ($r = -0.12$, $p < .05$) were associated with higher depressive symptoms. Because both meth-using MSM groups displayed higher free T3, we tested whether this mediated the associations of Meth/HIV group with greater depressive symptoms. These indirect effects were not significant.

Discussion

This study examined the association of meth use and HIV with thyroid function among MSM in South Florida, and explored its relationship with demographic, psychiatric, and physiological factors. Findings indicated a pattern of thyroid dysregulation among meth-using MSM, irrespective of HIV status. METH+HIV+ and METH+HIV– MSM displayed significantly higher levels of free T3 and had significantly greater odds of abnormal TSH compared to METH–HIV– men. Although higher free T3 was also associated with greater depressive symptoms, free T3 did not mediate greater depressive symptoms in either group of meth-using MSM. Taken together, findings demonstrate that regular screening of thyroid functioning should be considered in meth users, particularly those who present with depressive symptoms.

Informed by negative reinforcement models of addiction (Baker et al., 2004), depressive symptoms promote continued meth use to avoid withdrawal symptoms and serve as a potent trigger for relapse. There is some evidence that the association of meth use with greater depressive symptoms may be partially attributable to thyroid dysregulation (Li et al., 2013; Noureldeen et al., 2014), and this relationship is modified by HPA axis functioning (Zuloaga et al., 2013, 2015). In fact, both hypoactive and hyperactive thyroid function are associated with greater risk of depressive disorders (Hage & Azar, 2012). Although one prior study found no association of thyroid hormones with depressive symptoms in HIV-negative meth users (Li et al., 2013), we observed that higher free T3 was associated with greater depressive symptoms in meth-using MSM regardless of HIV status. Further clinical research is necessary to identify the bio-behavioral mechanisms underlying substantially elevated depressive symptoms among meth-using MSM to inform the development of novel intervention approaches.

Meth-using MSM also had greater odds of abnormal TSH compared to METH–HIV– men. These abnormal TSH levels were generally indicative of hyperactive pituitary functioning, but some meth-using MSM displayed evidence of hypoactive pituitary functioning. Because pituitary functioning is controlled by the hypothalamus, these results are consistent with prior research by our team and others, in which meth use was associated with HPA axis

dysregulation (Carrico et al., 2017; Li et al., 2013). Consistent with greater thyroid dysregulation, meth-using MSM were more likely to be normal weight and report greater depressive symptoms. These findings provide some support for hyperthyroidism among meth users, which is typically accompanied by weight loss and mood symptoms.

Results of this study must be viewed within certain limitations. First, meth use was assessed by self-report using an administered structured interview. Participants may have under-reported meth use or the use of other drugs, though the majority of meth abusers did report polysubstance use. In fact, neither the length of meth use, the quantity of use, nor an HIV-infected non-meth using group were assessed, all of which should be considered in future studies. Second, this cross-sectional study utilized an intact groups design to compare meth-using MSM to men who were not meth users. Longitudinal research that attempts to match meth users with non-users is needed to build upon these findings. Third, HIV treatment (highly active antiretroviral therapy) may have increased the probability of thyroid dysfunction but we observed comparable thyroid dysregulation in HIV-negative, meth-using MSM.

While most reports in substance using populations have investigated opiate and cocaine use, this study presented evidence of thyroid dysfunction in meth-using MSM. It was also observed that depression was significantly higher in meth abusers, which may be partially attributable to thyroid dysfunction in meth-using MSM. Future longitudinal studies should elucidate the bio-behavioral mechanisms whereby meth use could dysregulate thyroid functioning. Overall, results demonstrate the clear need for medical assessment of thyroid function in meth-using populations, particularly among those presenting with depression.

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Table 1

Sociodemographic, psychiatric and biomarker comparisons (N = 309)

	METH–HIV– n = 136 N (%)	METH+HIV+ n = 127 N (%)	METH+HIV– n = 46 N (%)	X²/F, <i>p</i>
Race/ethnicity				17.52, 0.002
Caucasian	12 (8.8%)	34 (26.8%)	13 (28.3%)	
African American	38 (27.9%)	33 (26.0%)	9 (19.6%)	
Hispanic and others	86 (63.2%)	60 (47.2%)	24 (52.2%)	
Monthly Personal Income (USD)				20.80, < 0.001
Less than \$500	44 (32.4%)	75 (59.1%)	26 (56.5%)	
\$500 or more	92 (67.6%)	52 (40.9%)	20 (43.5%)	
Body Mass Index (BMI)				23.47, < 0.001
Normal	34 (25.0%)	60 (47.6%)	25 (54.3%)	
Overweight	58 (42.6%)	47 (37.3%)	14 (30.4%)	
Obese	44 (32.4%)	19 (15.1%)	7 (15.2%)	
Polysubstance use disorder	3 (2.2%)	90 (70.9%)	32 (69.6%)	147.54, < 0.001
	Mean (SD)	Mean (SD)	Mean (SD)	
Age	35.26 (10.45) ^a	39.85 (8.39) ^{a,c}	33.22 (9.50) ^c	11.59, < 0.001
Childhood abuse	51.51 (11.67) ^{a,b}	70.51 (17.73) ^a	64.31 (16.02) ^b	53.29, < 0.001
Free T3 (log ₁₀)	0.36 (0.11) ^{a,b}	0.41 (0.13) ^a	0.43 (0.15) ^b	17.82, < 0.001 ^I
Free T4 (log ₁₀)	– 0.08 (0.08)	– 0.09 (0.08)	– 0.10 (0.08)	1.47, 0.48 ^I
TSH (log ₁₀)	0.08 (0.32)	0.12 (0.36)	0.10 (0.49)	2.96, 0.23 ^I
Depression (CES-D)	10.93 (6.89) ^{a,b}	28.42 (9.92) ^a	26.39 (10.20) ^b	143.52, < 0.001

T3, triiodothyronine; T4, thyroxine; TSH, thyroid stimulating hormone

^aMETH–HIV– versus Meth+HIV+, *p* < .05;^bMeth–HIV– versus METH+HIV–, *p* < .05;^cMeth+HIV+ versus METH+HIV–, *p* < .05^IKruskal Wallis tests were used for median comparison of groups and Chi square tests for differences in proportions

Table 2

Correlates of hypothalamic–pituitary–thyroid functioning (N = 309)

	Abnormal Free T3 AOR (95% CI)	Abnormal Free T4 AOR (95% CI)	Abnormal TSH AOR (95% CI)
METH/HIV Group			
METH–HIV– (reference)	–	–	–
METH+HIV+	1.99 (0.90–4.45)	0.99 (0.41–2.41)	4.51 (1.34–15.24) *
METH+HIV–	1.31 (0.47–3.63)	1.97 (0.69–5.59)	8.87 (2.46–32.00) **
Age	1.00 (0.98–1.03)	1.05 (1.02–1.09) **	0.98 (0.94–1.03)
Race/ethnicity			
Caucasian (reference)	–	–	–
African American	1.28 (0.56–2.92)	2.85 (1.26–6.42) *	0.26 (0.09–0.78) *
Hispanic/Latino	0.82 (0.38–1.76)	0.80 (0.37–1.75)	0.20 (0.08–0.52) **
Other Ethnic Minority	2.66 (0.75–9.45)	0	0.91 (0.19–4.38)
Body Mass Index (BMI)			
Normal (reference)	–	–	–
Overweight	1.69 (0.88–3.28)	1.40 (0.72–2.72)	0.57 (0.22–1.50)
Obese	2.12 (1.01–4.44) *	0.87 (0.38–1.97)	1.41 (0.49–4.04)
Childhood abuse	0.97 (0.95–0.99) **	0.99 (0.98–1.01)	1.00 (0.98–1.03)
Polysubstance use disorder	0.73 (0.34–1.58)	0.98 (0.44–2.18)	0.38 (0.15–0.95) *

T3, triiodothyronine; T4, thyroxine; TSH, thyroid stimulating hormone

*
 $p < .05$;**
 $p < .01$. [N.B.: hyperthyroid = low TSH and high T3 or T4]