BODY-MASS INDEX AND INFLAMMATION IN PEOPLE LIVING WITH HIV AND UNINFECTED CONTROLS

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BACKGROUND

In the present study we aimed to explore differences in inflammatory markers between people living with HIV (PLWH) and uninfected controls before and after stratification according to body-mass-index.

METHODS

710 well-treated PLWH and 86 uninfected controls where included from the Copenhagen Comorbidity in HIV infection (COCOMO) study. Study participants were stratified into overweight/obese and normo-/underweight according to their BMI (<25 and ≥ 25, respectively). Hypotheses were tested by linear and logistic regression analyses adjusted for age and sex.

RESULTS

PLWH had lower BMI compared to uninfected control (25.1 vs 26.2, p-value 0.004), but no difference in prevalence of overweight/obesity was found between the two groups (43% vs 53%, p-value 0.086). While no differences in IL-6, IL-10, IFN-γ and hs-CRP were found, PLWH had higher levels of TNF-alfa (7.9 (4.6) vs 6.7 (2.6), p-value 0.041) compared to uninfected controls. After stratification according to BMI and adjustment for confounders HIV infection was associated with excess risk of high levels of IL-6 (aOR 5.82 [1.69 – 20.05]) and IFN-γ (aOR 3.41 [1.01 – 11.46]) in normoweight/underweight,

Table 1 – Predictors of IL-6 and Interferon-γ levels in PLWH and uninfected controls after stratification according to BMI

	Interleukin-6			
	Normoweight/underweight		Overweight/obese	
	PLWH *Beta [95% CI]*	Uninfected controls *Beta [95% CI]*	PLWH *Beta [95% CI]*	Uninfected controls *Beta [95% CI]*
Age, per year	**0.06 [0.03;0.10]***	0.07 [-0.02;0.16]	**0.09 [0.06;0.12]***	0.04 [-0.03;0.11]
Sex, male vs female	0.34 [-0.64;1.32]	0.51 [-1.00;2.02]	-0.05 [-1.09;0.99]	-1.78 [-3.41;-0.16]
WHR, per 1 unit change	**5.69 [0.41;10.97]***	2.95 [-8.24;14.15]	0.34 [-4.55;5.23]	3.56 [-9.33;16.46]
Smoking, yes vs no	**1.38 [0.69;2.07]***	0.89 [-1.89;3.66]	0.53 [-0.26;1.31]	-0.38 [-2.70;1.93]

	Interferon-γ			
	Normoweight/underweight		Overweight/obese	
	PLWH *Beta [95% CI]*	Uninfected controls *Beta [95% CI]*	PLWH *Beta [95% CI]*	Uninfected controls *Beta [95% CI]*
Age, per year	-0.01 [-0.03;0.00]	0.00 [-0.02;0.02]	-0.01 [-0.07;0.05]	-0.01 [-0.05;0.02]
Sex, male vs female	-0.18 [-0.70;0.33]	-0.41 [-0.77;-0.05]	0.38 [-1.42;2.17]	-0.65 [-1.48;0.17]
WHR, per 1 unit change	0.16 [-2.63;2.96]	1.31 [-1.33;3.95]	4.14 [-4.41;12.68]	**8.64 [2.11;15.17]***
Smoking, yes vs no	-0.09 [-0.45;0.27]	-0.16 [-0.81;0.48]	-0.17 [-1.54;1.21]	-0.69 [-1.87;0.48]

*, p-value < 0.05

This result was not consistent among overweight/obese individuals (aOR 2.03 [0.91 – 4.55] and aOR 2.46 [0.93 – 6.49], respectively). When restricting the analyses to normoweight/underweight individuals, age, smoking and waist-to-hip ratio were associated with higher concentrations of IL-6 in PLWH, but not in uninfected controls (Table 1).

CONCLUSIONS

In the present study, HIV was associated with higher levels of IL-6 and IFN-γ in normoweight/underweight but not in overweight/obese individuals. Interestingly, waist-to-hip ratio was a predictor of higher IL-6 in normoweight PLWH and not in uninfected controls. Taken together, our results may suggest a pivotal role for adipose tissue deposition in the early establishment of a pro-inflammatory milieu, even in individuals not clinically overweight or obese. Given the well-known association of systemic inflammation with non-AIDS associated cardio metabolic comorbidities, our results may suggest the need of more intensive monitoring for comorbidities in a subgroup of PLWH otherwise described at low risk.

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