

RESEARCH ARTICLE

## Association Between Hearing Loss Suspect with Diabetes and Arterial Hypertension Risk Under Different Body Composition Phenotypes: Results from the Chilean National Health Survey 2016-17

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### Abstract

Purpose: 1) To characterize the glucose and blood pressure control in HLS adult participants based on four body composition phenotypes models from the Chilean National Health Survey 2016-17. 2) to associate the HLS and body composition phenotypes (based on calf and waist circumference [WC]) with arterial hypertension and diabetes markers. Method: A quantitative cross-sectional study based on the Chilean National Health Survey 2016-17, reporting adults with/without HLS based on four different phenotypes was carried out by the epidemiological group of the Institute of Rehabilitation and Exercise Sciences of the Universidad Andres bello, Chile (ICER-UNAB). Groups were as follows; Low skeletal muscle [SMM] and high waist circumference [WC] (Lsmm-Hwc,  $n=278$ ), Low SMM and low WC (Lsmm-Lwc,  $n=479$ ), High SMM and high WC (Hsmm-Hwc,  $n=2140$ ), and High SMM and low WC (Hsmm-Lwc,  $n=2709$ ). The primary outcomes were fasting plasma glucose, systolic, and diastolic blood pressure. Findings: In systolic blood pressure, there were significant differences between each Lsmm-Hwc (150 vs. 123 mmHg), Lsmm-Lwc (140 vs. 123 mmHg), and Hsmm-Hwc vs. the Reference group (145 vs. 123 mmHg, all  $P<0.0001$ ). Systolic blood pressure reported significant Group x HLS interaction  $F(3.33)$ ,  $P=0.019$ , ES 0.004. In diastolic blood pressure, there were significant differences between each Lsmm-Hwc (74 vs. 72 mmHg) and Hsmm-Hwc vs. the Reference group (76 vs. 72 mmHg, both  $P<0.0001$ ). Diastolic blood pressure reported significant Group x HLS interaction  $F(4.49)$ ,  $P=0.004$ , ES 0.005. Chilean adults with HLS, low SMM and high waist circumference shows a higher systolic and diastolic blood pressure.

### Keywords

Body Composition, Hearing Loss, Quality of Life, Diabetes, Arterial Hypertension

## INTRODUCTION

Considering the different and potential causes of hearing loss (HL) in adults, the natural ageing of the auditory system (Tang, Tran,

Dawes, & Gopinath, 2023) and prolonged exposure to high noises are commonly the two main factors described in the HL process (Elshaer, Meleis, & Mohamed, 2023). Worryingly, more than 65% of people over 60 years old suffer from

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some degree of hearing loss (i.e., independently of their congenital, permanent, bilateral, or unilateral HL cause); however, the prevalence increases dramatically in 15% of those aged 60, and 80% among those aged  $\geq 80$  years old (Goman & Lin, 2016). It has been described that 62.7 million Americans have some degree of HL (i.e., moderate, or high grade) (WHO, 2021b); however, it is increased by  $\sim 10\%$  in  $>15$  aged population that suffer from any disability (i.e., from sensory, hearing, or visual disability). Indeed, literature has reported that HL disability represents  $\sim 8.2\%$  of total disabilities (WHO, 2021b).

Independently of the HL origin and prevalence, together with the physical health disabilities that HL is related to, other health-related quality-of-life (QoL) detriments are also characteristic of adults with some degree of HL or hearing loss suspect (i.e., a self-report of some degree of hearing loss without a clinical diagnosed). Moreover, other conditions such as lower postural stability (Foster, Williams, Timmer, & Brauer, 2022), cognitive impairment, dementia (Azeem et al., 2023; Tamblay et al., 2023), depression (Baiduc, Sun, Berry, Anderson, & Vance, 2023), and poor QoL have been associated with populations with HLS as well (Dillard et al., 2023).

Reductions in the capacity for adhering to and maintaining daily living activities that could protect cardiometabolic health (i.e., decreased capacity to maintain regular physical activity such as walking or running, to associate with other peers by limited social possibilities associated with HLS) are common aspects related to poor QoL (Goodwin, Hogervorst, Hardy, Stephan, & Maidment, 2023; Kuo, Di, Ferrucci, & Lin, 2021). Furthermore, the reduction in physical activity levels in adults with HLS is usually related to more time in sedentary activities, and these populations could face less possibility the cardiometabolic conditions usually associated with the ageing process, such as diabetes and HTN (Goodwin et al., 2023). Thus, when adults declare HLS, they could probably be at major risk of modifying their body composition (i.e., increasing body fat, and reducing their skeletal muscle mass) and thus more at risk for acquiring comorbidities such as being overweight or obesity condition (Curhan, Eavey, Wang, Stampfer, & Curhan, 2013). So far, a previous study involving ( $n=62.421$ ) women reported that adults with HL

showed a higher body mass index (BMI) and higher waist circumference (WC) characteristically; meanwhile, peers that walk at least 2 h per week have shown an inverse association with a minor HL prevalence (Curhan et al., 2013). Therefore, physical inactivity (i.e., do not adhere to international physical activity guidelines of at least 150 to 300 min of low-to-moderate PA per week), or 75 to 150 min of vigorous PA per week (Tsao et al., 2023)) and sedentary time usually increase adiposity (i.e., by BMI and body fat increases) (Croll et al., 2019), contributing to skeletal muscle mass (SMM) decrease and SMM loss (sarcopenia), in addition to lower glucose and blood pressure control, leading for diabetes and arterial hypertension risk (Park et al., 2022).

So far, changes in body composition have been linked with HLS in the adult population, which leads to a greater risk of suffering from cardiometabolic diseases such as diabetes and arterial hypertension (HTN). Likewise, Samocha-Bonet et al. (2021) highlight that in older adults diagnosed with diabetes, the incidence of HL increases twice. Moreover, several studies have also reported a significant association between HL and a higher prevalence of diabetes and HTN (Helzner et al., 2011; McKee, Stransky, & Reichard, 2018; Samocha-Bonet, Wu, & Ryugo, 2021). Several investigations (Bener et al., 2017; Gioacchini et al., 2023; Meneses-Barriviera et al., 2018) have reported epidemiological data about adults with HLS and the association with cardiometabolic diseases; however, no studies have established these associations in Chilean population, especially the relation of HLS with diabetes and HTN risk.

On the other hand, it is interesting to mention a recent preclinical study in animal models from (Park et al., 2020) that suggests the acceleration of the ageing process (by increasing oxidative-stress-induced mitochondrial dysfunction, promoting hypoxia or increasing the exposure to high-fat diet) was significantly associated with high HL risk.

Concerning this, characterize the different body composition phenotypes (i.e., high, or low SMM, by calf circumference; and high or low body fat, by WC or their combinations) and their association with diabetes and HTN markers in the adult Chilean population with HLS would

contribute to clinical practice and prevention is little information regarding the cardiometabolic risk in terms of HTN and diabetes prevalence in the population with HLS, being relevant to describe the characteristics and lifestyle of the population with HLS where their body composition phenotypes could play a role in preventing additional or major comorbidities, and above considering the role of a low SMM and higher body in the worse of the glucose control (i.e., diabetes), and impairment of the cardiovascular health (i.e., HTN).

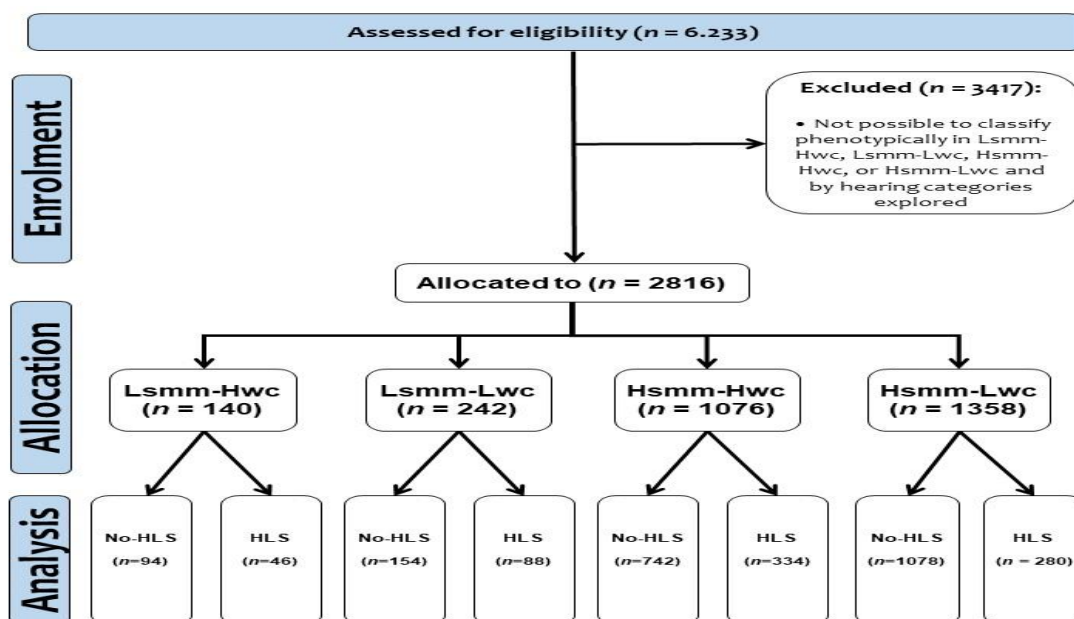
Therefore, this study aims: 1) to characterize the glucose and blood pressure control in HLS adult participants based on four body composition phenotypes models from the Chilean National Health Survey 2016-17 (NHS16-17) and 2) to associate HLS and body composition phenotypes (based on calf and waist circumference) with arterial hypertension and diabetes markers.

**MATERIALS AND METHODS**

This cross-sectional study is based on the NHS16-17, a prevalence, multi-stage, and representative study developed at home, using random, stratified-by-conglomerates methods, that include urban and rural areas (Minsal, 2017).

strategies for improving QoL. Additionally, there **Participants**

Considering the total NHS16-17 sample of (n=6.233) participants, the present study included (n=5.632) participants that were associated by each phenotype-modelled group and by the categories of “no hearing loss suspect” (No-HLS) and “hearing loss suspect” (HLS). The final sample was then determined according with four different body composition phenotypes using each SMM and CC variations, as follows; Low SMM and high WC (Lsmm-Hwc, n=140 [category No-HLS, n=94; HLS, n=46]), Low SMM and Low WC (Lsmm-Lwc, n=242 [category No-HLS, n=154; HLS, n=88]), High SMM and High WC (Hsmm-Hwc, n=1076 [category No-HLS, n=742; HLS, n=334]), and High SMM and Low WC (Hsmm-Lwc, n=1358 [category No-HLS, n=1078; HLS, n=280]). The study protocol was approved by the Ethical Committee of the Escuela de Medicina de la Pontificia Universidad Católica de Chile (16-019), and all participants signed an informed consent, date: ENS16-17. The study design can be seen in (Figure 1). The research strictly adhered to the ethical principles of the Declaration of Helsinki, prioritizing participant's rights and well-being in design, procedures, and confidentiality measures.



**Figure 1.**The study design

**Body composition phenotypes**

The participants were grouped by four phenotypes according to a different SMM level (i.e., based on the ‘calf circumference’ outcome)

and adiposity (i.e., based on the ‘waist circumference’ outcome) that are reported in the NHS16-17.

The four groups of phenotypes (Lsmm-Hwc, Lsmm-Lwc, Hsmm-Hwc, Hsmm-Lwc) were modelled using the cut-off point of 34 cm to ‘calf circumference’ for men, and 33 cm for adult women that have demonstrated high sensitivity (80.0%) and specificity (84.6%) (Pagotto, Santos, Malaquias, Bachion, & Silveira, 2018). The WC was categorized as ‘higher’ WC (men  $\geq 90$  cm, women  $\geq 80$  cm), or ‘low’ WC (men  $< 89$  cm, women  $< 79$  cm), as previously used (Rolland et al., 2003). For both CC and WC outcomes, a professional nurse used an inextensible tape for the measurement applied at each participant’s home. On the other hand, the No-HLS or HLS condition was categorized using the data population that reported suffering (or not) of HLS, using the question included in the NHS16-17 as follows; “Do you consider that you normally hear in both ears?”.

#### **Diabetes and arterial hypertension markers (main outcomes)**

Fasting plasma glucose (FPG) and glycated haemoglobin were measured in fasting conditions (i.e., 8 h) by professional nursing, similar to those reported in previous studies (Álvarez et al., 2023). To detect the diabetes risk, there was reported the odds ratio (OR) statistical parameter to detect the risk of suffering or not some condition, such as diabetes and HTN risk, by the categorization of the question; ‘diabetes suspects in fasting state’, with categorical answers ‘Yes’ or ‘Not’, included in the NHS16-17.

On the other hand, to the HTN risk, the systolic (SBP) and diastolic (DBP) were measured in the left arm three times, and the average of these attempts was registered. We used the American Heart Association 2018 blood pressure categorization; ‘Normal BP’ was defined as SBP/DBP less than 120/80 mmHg, ‘elevated blood pressure’ (Ele) as SBP/DBP 120-129/80 mmHg, ‘stage 1 HTN’ as SBP/DBP 130-139/80-89 mmHg, and ‘stage 2 HTN’ as SBP/DBP  $\geq 140/90$  mmHg (Whelton et al., 2018). These measurements were carried out by an automatic monitor (OMRON™, model HEM 7114, Tokyo, Japan) similar to previous studies of the NHS16-17 (Petermann, Duran, et al., 2017), and were applied by professional nursing at-home conditions. In addition, to detect the HTN risk, we also reported the OR statistical parameters, and used the question; ‘Arterial hypertension suspects’,

with categorical answers ‘Yes’ or ‘Not’, included in the NHS16-17.

#### **Secondary cardiometabolic risk factors (Secondary outcomes)**

Total cholesterol (Tc), low-density lipid cholesterol (LDL-c), high-density lipid cholesterol (HDL-c), and plasma triglycerides (Tg) were measured and classified following the National Cholesterol Education Program NCEP ATP-III criteria (NCEP, 2002). The 25-OH vitamin D<sub>2</sub>+D<sub>3</sub> outcome was included as a mineral content marker, and gamma (GGT) and pyruvic glutamyl transferase (PGT) were analyzed as non-alcoholic fatty liver disease markers. Free thyroxine and thyroid stimulating hormone (TSH) were analyzed as markers of ‘hypothyroidism’, C-Reactive protein was included as a marker of ‘inflammation’, and finally, microalbuminuria was included as a marker of kidney damage.

The weight was measured by a digital electronic scale OMRON™, model HN 289 (OMRON Corporation, Kyoto, Japan), (sensitivity of 100 g, maximum weight capacity of ~150 kg), where height and waist circumference were assessed by an inextensible tape, similar as previously (Concha-Cisternas et al., 2023). The BMI was calculated using weight and height information from international institutions (WHO, 2000).

#### **Cardiovascular risk score estimation**

The cardiovascular risk was categorized by scale punctuation in ‘low’ (0-4 points), ‘moderate’ (5 to 9 points), and ‘high cardiovascular risk’ ( $\geq 10$  points) using the metabolic syndrome outcomes (SBP, DBP, FPG, HDL-c, and Tg), including tobacco habit, alcohol consumption, dyslipidemia, sleep patterns, as well by the three questions included as follows; a) in the self-report on acute myocardial infarction ‘Has a doctor or physician ever told you had or suffered a heart attack? Being the prevalence from those who answered “Yes”’; b) the question for the self-reported prevalence of stroke “Has a doctor or physician ever told you had or suffered a stroke? or had or suffered a stroke or cerebral thrombosis (or stroke)?”, and c) the question about the self-reported prevalence of peripheral venous disease “Has a doctor or physician ever told you had or suffered from peripheral vascular disease or to the arteries in your legs?” (MINSAL, 2018). However, we reported only the risk of suffering from ‘moderate’ and high CVR’ in this study.

### Statistical analysis

Data for continuous outcomes are shown as mean and (95%CI), and for categorical outcomes as frequency ( $n=$ ) and (%), percentage. The normality was tested using the Shapiro-Wilk test. For continuous outcomes, the interaction of the four phenotypes groups (Lsmm-Hwc, Lsmm-Lwc, Hsmm-Hwc, and Hsmm-Lwc) with HLS categories was tested using univariant analyses ANOVA (Groups; HLS; and Groups x HLS). Moreover, using multinomial logistic regression, we calculated the risk for suffering from diabetes, HTN, MetS, 'moderate' and 'high' cardiovascular risk by the OR and showed the information as mean and (95% CI). The Wald Chi-square was reported and the pseudo-McFadden  $R^2$  were reported for predicting each dependent outcome. Additionally, we calculated the effect size (ES) using Cohen's  $d$  test (Hopkins, Marshall, Batterham, & Hanin, 2009) corrected for small samples (<20 subjects) (Hedges & Olkin, 2014), with threshold values at 0.20, 0.60, 1.2, and 2.0 for

small, moderate, large, and very large effect sizes, respectively. These analyses were adjusted by geographic area, region, sex, and age. All statistical analyses were developed using the SPSS™ software 25 version for Windows (IBM SPSS Inc., Chicago, IL, USA).

### RESULTS

The general characteristics of No-HLS vs. HLS group revealed significant differences in outcomes age (*diff.* 10.4 y,  $P<0.0001$ ), height (*diff.* 2 cm,  $P<0.0001$ ), weight (*diff.* 2.1 kg,  $P=0.003$ ), FPG (*diff.* 3.4 mg·dL), SBP (*diff.* 6 mmHg,  $P<0.0001$ ), PGT (*diff.* 1.7 UI·L,  $P=0.035$ ) microalbuminuria (*diff.* 1.85 mg·dL,  $P=0.007$ ) and  $PA_{VI}$  (*diff.* 6.2 min·week,  $P=0.002$ ) (Table 1). No significant differences were detected between No-HLS vs. HLS group in outcomes BMI, WC, CC, HbA1c, DBP, Tc, LDL-c, HDL-c, Tg, vitamin D2+D3, GGT, PGT, free thyroxine, TSH, CRP,  $PA_{MI}$ ,  $PA_{LI}$ , handgrip muscle strength (Table 1).

**Table 1.** General characteristics of adult participants of the Chilean national health survey 2016-17.

Outcomes ( $n =$ )	No-HLS 4929	HLS 1304	No-HLS vs. HLS
Age (y)	46.7 (46.1; 47.2)	57.1 (56.1; 58.1)	<b><math>P&lt;0.0001</math></b>
Height (cm)	160.0 (159.7; 160.2)	158.0 (157.5; 158.6)	<b><math>P&lt;0.0001</math></b>
Weight (kg)	74.0 (73.5; 74.4)	71.9 (71.0; 72.8)	<b><math>P=0.003</math></b>
BMI (kg·m <sup>2</sup> )	27.2 (26.9; 27.4)	27.0 (26.6; 27.4)	$P=0.451$
WC (cm)	93.6 (93.1; 94.0)	93.9 (93.1; 94.7)	$P=0.535$
CC (cm)	35.1 (34.8; 35.4)	34.8 (34.3; 35.2)	$P=0.191$
FPG (mg·dL)	99.3 (98.2; 100.4)	102.7 (100.5; 104.9)	<b><math>P=0.007</math></b>
HbA1c (%)	6.3 (6.2; 6.4)	6.5 (6.3; 6.6)	$P=0.098$
SBP (mmHg)	126 (125; 126)	132 (131; 133)	<b><math>P&lt;0.0001</math></b>
DBP (mmHg)	75 (74; 75)	74 (74; 75)	$P=0.773$
Tc (mg·dL)	181.1 (179.6; 182.6)	182.4 (179.6; 185.2)	$P=0.422$
LDL-c (mg·dL)	104.6 (103.4; 105.8)	105.5 (103.1; 107.8)	$P=0.529$
HDL-c (mg·dL)	47.7 (47.2; 48.1)	48.2 (47.3; 49.1)	$P=0.311$
Tg (mg·dL)	144.0 (140.6; 147.3)	143.7 (137.3; 150.1)	$P=0.934$
D2+D3 (ng·mL)	19.8 (19.5; 20.2)	19.3 (18.7; 19.9)	$P=0.158$
GGT (UI·L)	32.0 (30.2; 33.8)	34.4 (30.9; 37.8)	$P=0.229$
PGT (UI·L)	25.3 (24.6; 26.0)	23.6 (22.3; 25.0)	<b><math>P=0.035</math></b>
TSH (ng·dL)	3.97 (3.46; 4.47)	4.14 (3.20; 5.09)	$P=0.746$
FT (ng·dL)	1.20 (1.18; 1.21)	1.20 (1.17; 1.23)	$P=0.806$
C-RP (mg·L)	0.45 (0.38; 0.52)	0.40 (0.29; 0.51)	$P=0.431$
Microalb	2.86 (2.21; 3.50)	4.71 (3.52; 5.89)	<b><math>P=0.007</math></b>
$PA_{VI}$ (min·week)	3.5 (2.8; 4.2)	6.1 (4.6; 7.6)	<b><math>P=0.002</math></b>
$PA_{MI}$ (min·week)	4.0 (3.5; 4.6)	4.2 (3.1; 5.3)	$P=0.749$
$PA_{LI}$ (min·week)	12.7 (12.2; 13.2)	12.2 (11.2; 13.2)	$P=0.431$
HGS (kg)	52.1 (46.8; 57.5)	59.5 (52.3; 66.7)	$P=0.108$

Data are shown as mean and (95% CI). Groups are described as; (No-HLS) No hearing loss suspect. (HLS) Hearing loss suspect. (BMI) Body mass index. (WC) Waist circumference. (CC) Calf circumference. (FPG) Fasting plasma glucose. (HbA1c) Glycated hemoglobin. (SBP) Systolic and (DBP) Diastolic blood pressure. (Tc) Total cholesterol. (LDL-c) Low-density lipoprotein. (HDL-c) High-density lipoprotein. (Tg) Triglycerides. (D2+D3) Vitamin D2+D3. (GGT) Gamma glutamil transferase. (PGT) Piruvate glutamil transferase. (TSH) Tirostimulant hormone. (FT) Free thyroxine. (C-RP) C-Reactive protein. (Microalb) Microalbuminuria. (PA<sub>VI</sub>) Vigorous-intensity physical activity. (PA<sub>Mi</sub>) Moderate-intensity physical activity. (PA<sub>L</sub>) Light-intensity physical activity. (HGS) Handgrip muscle strength. Bold values denote significant differences by groups at  $P \leq 0.05$ .

There were significant differences in FPG between each Lsmm-Hwc (112.5 vs. 93.6 mg·dL,  $P < 0.0001$ ), Lsmm-Lwc (95.8 vs. 93.6 mg·dL,  $P < 0.021$ ), and Hsmm-Hwc vs. the Ref. group (112.3 vs. 93.6 mg·dL,  $P < 0.0001$ ) (Figure 2A). Significant Group interaction was reported in FPG  $F(22.10)$ ,  $P = 0.015$ , ES 0.957 (Figure 2A). In HbA1c, no significant interactions were found (Figure 2B). In SBP, there were significant differences between each Lsmm-Hwc (150 vs. 123 mmHg), Lsmm-Lwc (140 vs. 123 mmHg),

and Hsmm-Hwc vs. the Ref. group (145 vs. 123 mmHg, all  $P < 0.0001$ ) (Figure 2C). SBP reported significant Group x HLS interaction  $F(3.33)$ ,  $P = 0.019$ , ES 0.004 (Figure 2C). In DBP, there were significant differences between each Lsmm-Hwc (74 vs. 72 mmHg) and Hsmm-Hwc vs. the Ref. group (76 vs. 72 mmHg, both  $P < 0.0001$ ) (Figure 2D). DBP reported significant Group x HLS interaction  $F(4.49)$ ,  $P = 0.004$ , ES 0.005 (Figure 2D).

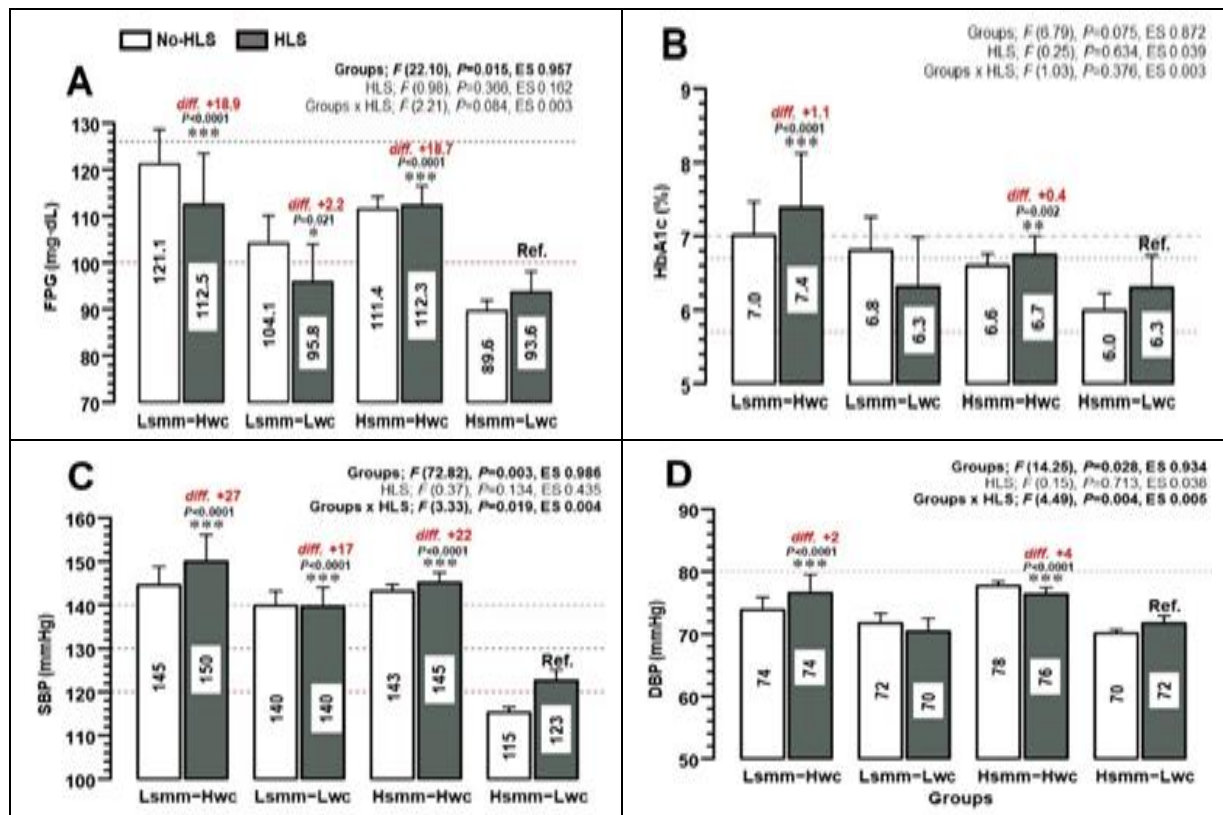
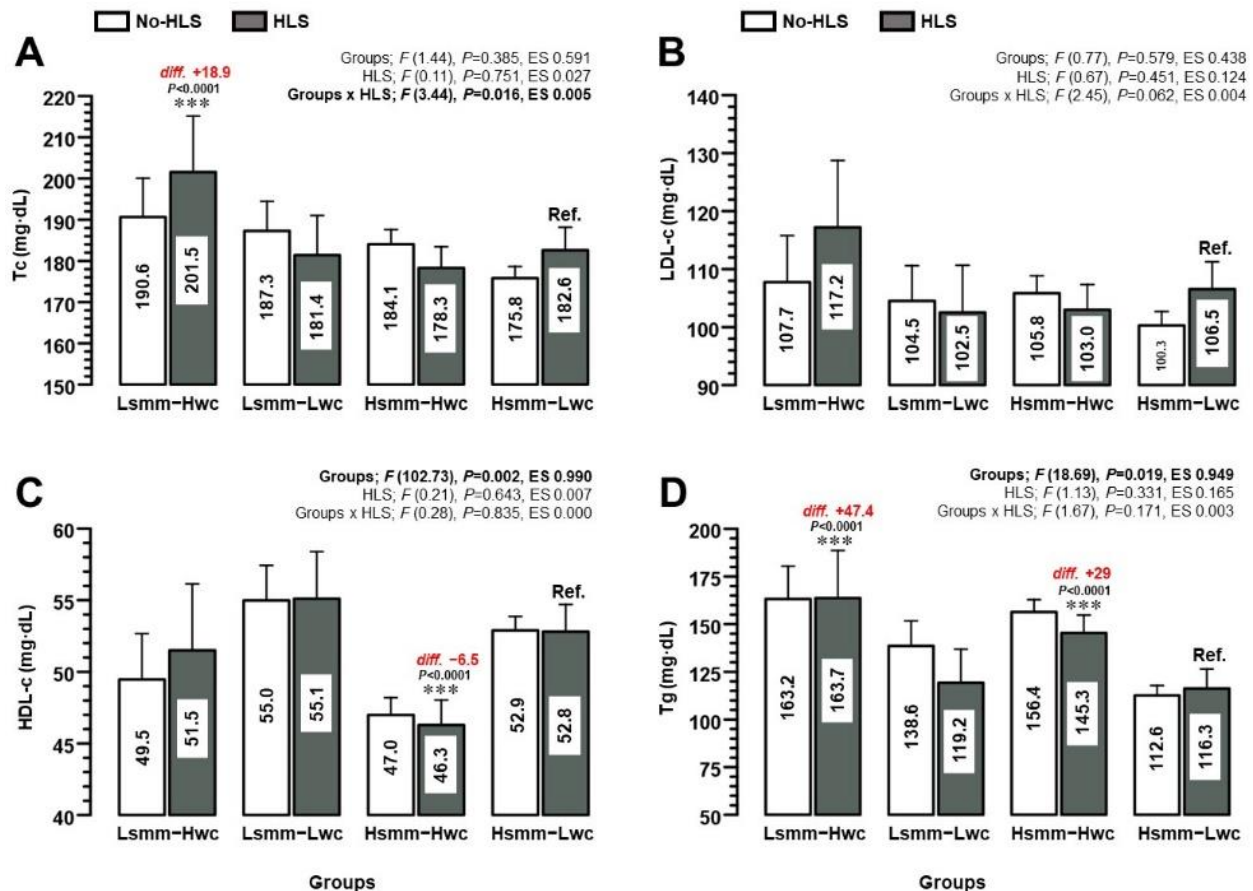


Figure 2. Diabetes (A, B) and arterial hypertension (C, D) markers in Chilean adult participants of the National Health Survey 2016-17, described by four body composition phenotypes and by a different condition of hearing. Groups are described as low-skeletal muscle mass and high waist circumference phenotypical model (Lsmm-Hwc), low-skeletal muscle mass and low waist circumference phenotypical model (Lsmm-Lwc), high-skeletal muscle mass and high waist circumference phenotypical model (Hsmm-Hwc), and high-skeletal muscle mass and low waist circumference phenotypical model (Hsmm-Lwc), Reference group (Ref.). Categories are described as: (HLS) hearing loss suspect. (No-HLS) No hearing loss suspect. Outcomes are described as; (FPG) Fasting plasma glucose. (HbA1c) Glycated hemoglobin. (SBP) Systolic blood pressure. (DBP) Diastolic blood pressure. (diff.) Differences vs. Ref. group. (\*) Denotes significant difference vs. Ref. at  $P < 0.05$ . (\*\*) Denotes significant difference vs. Ref. at  $P < 0.01$ . (\*\*\*) Denotes significant difference vs. Ref. at  $P < 0.0001$ .

In the lipid profile outcomes, there were significant differences in Tc between Lsmm-Hwc vs. the Ref. group (201.5 vs. 182.6 mg·dL,  $P<0.0001$ ) (Figure 3A). A significant Group x HLS interaction was reported in Tc  $F(3.44)$ ,  $P=0.016$ , ES 0.005 (Figure 3A). In LDL-c, no significant interactions were found (Figure 3B). In HDL-c, there were significant differences between Hsmm-Hwc vs. the Ref. group (46.3 vs. 52.8

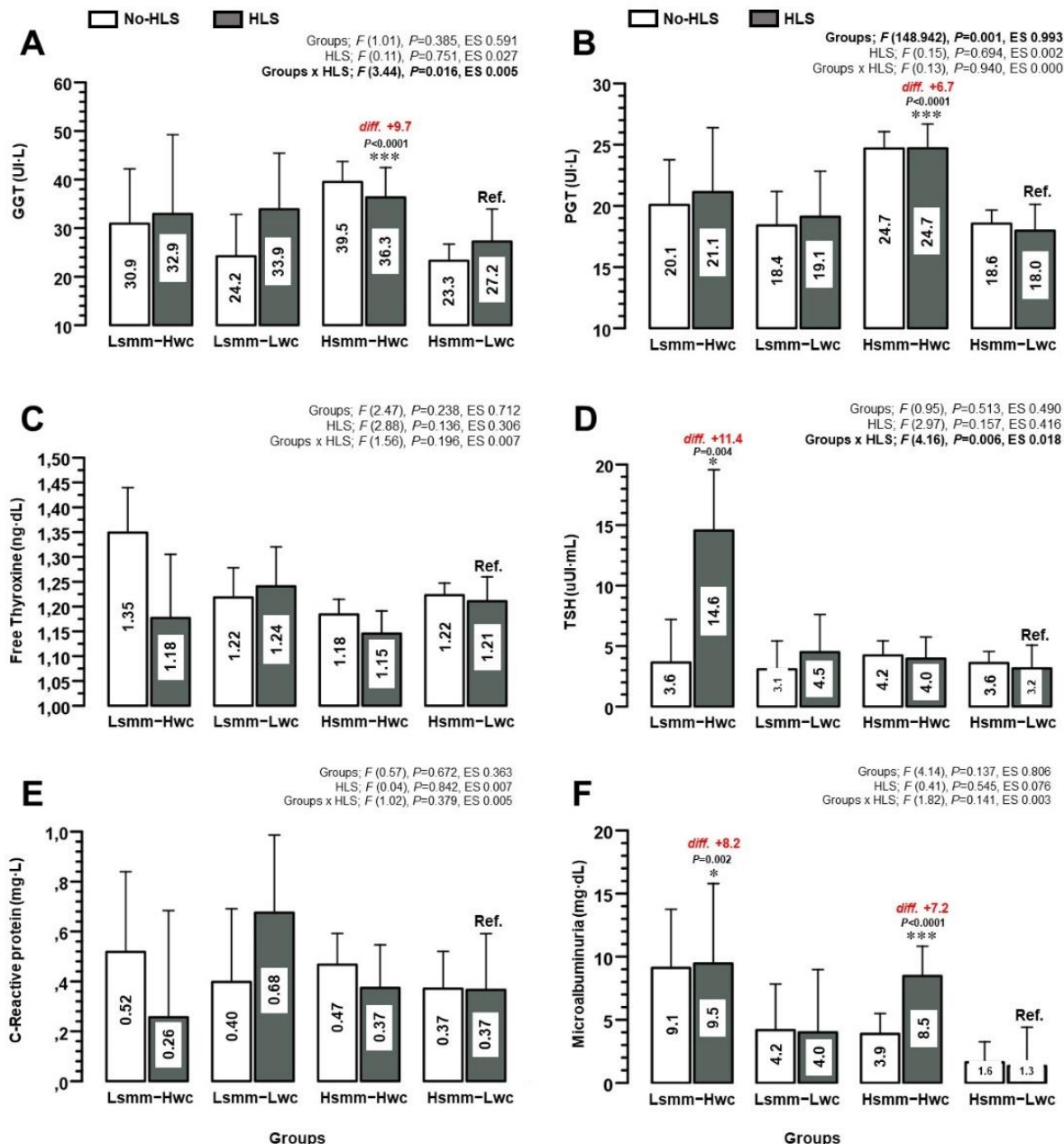
mg·dL,  $P<0.0001$ ) (Figure 3C). Significant Group interaction was reported in HDL  $F(3.44)$ ,  $P=0.016$ , ES 0.005 (Figure 3C). In Tg, there were significant differences between Lsmm-Hwc (163.7 vs. 116.3), and Hsmm-Hwc vs. the Ref. group (145.3 vs. 116.3 mg·dL,  $P<0.0001$ ) (Figure 3D). Significant Group interaction was reported in Tg  $F(18.69)$ ,  $P=0.019$ , ES 0.949 (Figure 3D).



**Figure 3.** Hypercholesterolemia/dyslipidaemia markers in Chilean adult participants of the National Health Survey 2016-17, described by four body composition phenotypes and by a different condition of hearing. Groups are described as: low-skeletal muscle mass and high waist circumference phenotypical model (Lsmm-Hwc), low-skeletal muscle mass and low waist circumference phenotypical model (Lsmm-Lwc), high-skeletal muscle mass and high waist circumference phenotypical model (Hsmm-Hwc), and high-skeletal muscle mass and low waist circumference phenotypical model (Hsmm-Lwc), Reference group (Ref.). Categories are described as HLS. (No-HLS) No hearing loss suspect. Outcomes are described as; (Tc) Total cholesterol. (LDL-c) Low-density lipoprotein. (HDL-c) High-density lipoprotein. (Tg) Triglycerides. (diff.) Differences vs. Ref. group. (\*\*\*) Denotes significant difference vs. Ref. at  $P<0.0001$ .

There were significant differences in GGT between Hsmm-Hwc vs. the Ref. group (14.6 vs. 3.2 UI·L,  $diff. +11.4$  UI·L,  $P=0.004$ ) (Figure 4D). A significant Group x HLS interaction was reported in TSH  $F(3.44)$ ,  $P=0.016$ , ES 0.005 (Figure 4A). There were significant differences in PGT between Hsmm-Hwc vs. the Ref. group (24.7 vs. 18.0 UI·L,  $diff. +6.7$  UI·L,  $P<0.0001$ )

(Figure 4B). Significant Group interaction was reported in PGT  $F(148.942)$ ,  $P=0.001$ , ES 0.993 (Figure 4B). There were significant differences in TSH between Lsmm-Hwc vs. the Ref. group (36.3 vs. 27.2 UI·L,  $diff. +9.7$  UI·L,  $P<0.0001$ ) (Figure 4D). A significant Group x HLS interaction was reported in GGT  $F(4.16)$ ,  $P=0.006$ , ES 0.018 (Figure 4D).



**Figure 4.** Non-alcoholic fatty liver disease (A, B), hypothyroidism (C, D), inflammation (E), and kidney disease markers (F) in Chilean adult participants of the National Health Survey 2016-17, described by four body composition phenotypes and by a different condition of hearing. Groups are described as low-skeletal muscle mass and high waist circumference phenotypical model (Lsmm-Hwc), low-skeletal muscle mass and low waist circumference phenotypical model (Lsmm-Lwc), high-skeletal muscle mass and high waist circumference phenotypical model (Hsmm-Hwc), and high-skeletal muscle mass and low waist circumference phenotypical model (Hsmm-Lwc), Reference group (Ref.). Categories are described as HLS. (No-HLS) No hearing loss suspect. Outcomes are described as; (GGT) Gamma glutamyl transferase. (PGT) Pyruvic glutamyl transferase. (TSH) Thyroid-stimulating hormone. (diff.) Differences vs. Ref. group. (\*) Denotes significant difference vs. Ref. at  $P<0.05$ . (\*\*\*) Denotes significant difference vs. Ref. at  $P<0.0001$ .

For diabetes suspect, there was a significant association between models 1 ( $\beta$  0.251,  $P=0.024$ ; OR 1.28), model 2 ( $\beta$  1.952,  $P<0.0001$ ; OR 7.03), model 3 ( $\beta$  1.315,  $P<0.0001$ ; OR 3.72), and model 4 ( $\beta$  1.917,  $P<0.0001$ ; OR 6.79) with diabetes diagnosed (Table 2). For HTN suspect, there was a significant association between models 1 ( $\beta$  0.367,  $P<0.0001$ ; OR 1.44), model 2 ( $\beta$  2.436,  $P<0.0001$ ;

OR 11.43), model 3 ( $\beta$  1.838,  $P<0.0001$ ; OR 6.28), and model 4 ( $\beta$  2.443,  $P<0.0001$ ; OR 11.50) with HTN diagnosed (Table 2). On the other hand, for ‘Hypercholesterolemia’ risk, there was a significant association between models 2 ( $\beta$  0.708,  $P<0.001$ ; OR 2.03), model 3 ( $\beta$  0.484,  $P=0.006$ ; OR 1.61), and model 4 ( $\beta$  0.319,  $P=0.004$ ; OR 1.37) (Table 2). For ‘MetS’ risk, there was a



significant association between models 2 ( $\beta$  2.268,  $P<0.0001$ ; OR 9.65), model 3 ( $\beta$  0.667,  $P<0.0001$ ; OR 1.94), and model 4 ( $\beta$  2.484,  $P<0.0001$ ; OR 11.9) (Table 2). For ‘High CVR’, there was a significant association between models 1 ( $\beta$  0.483,  $P=0.001$ ; OR 1.62), model 2 ( $\beta$  3.072,  $P<0.0001$ ;

OR 21.59), model 3 ( $\beta$  1.922,  $P<0.0001$ ; OR 6.83), and model 4 ( $\beta$  2.962,  $P<0.0001$ ; OR 19.34) (Table 2). Finally, for ‘NAFLD’ risk, there was a significant association between model 2 ( $\beta$  0.503,  $P=0.023$ ; OR 1.65), and model 4 ( $\beta$  0.533,  $P<0.0001$ ; OR 1.70) (Table 2).

**Table 2.** Multinomial logistic regression with odds ratios by each phenotype group and according to the risk for suffering different cardiometabolic conditions.

Outcomes	$\beta$	SE	Wald	McFadden Pseudo R <sup>2</sup>	OR (95%CI)	p-value
<b>‘Diabetes’</b>						
Model 0: No-HL or Hsmm-Lwc	-	-	-	-	<b>1.00 ( Ref. )</b>	-
Model 1: ‘HL’	0.251	0.111	5.075	0.115	1.28 (1.03; 1.59)	<b>P=0.024</b>
Model 2: Lsmm-Hwc	1.952	0.213	84.262		7.03 (4.64; 10.67)	<b>P&lt;0.0001</b>
Model 3: Lsmm-Lwc	1.315	0.197	44.424		3.72 (2.53; 5.48)	<b>P&lt;0.0001</b>
Model 4: Hsmm-Hwc	1.917	0.129	219.836		6.79 (5.27; 8.75)	<b>P&lt;0.0001</b>
<b>‘HTN’</b>						
Model 0: ‘No-HL’ or ‘Hsmm-Lwc’	-	-	-	-	<b>1.00 ( Ref. )</b>	-
Model 1: ‘HL’	0.367	0.101	13.208	0.218	1.44 (1.18; 1.76)	<b>P&lt;0.0001</b>
Model 2: Lsmm-Hwc	2.436	0.206	139.958		11.43 (7.63; 17.11)	<b>P&lt;0.0001</b>
Model 3: Lsmm-Lwc	1.838	0.150	150.897		6.28 (4.68; 8.43)	<b>P&lt;0.0001</b>
Model 4: Hsmm-Hwc	2.443	0.098	622.055		11.50 (9.49; 13.94)	<b>P&lt;0.0001</b>
<b>‘Hipercholesterolemia’</b>						
Model 0: ‘No-HL’ or ‘Hsmm-Lwc’	-	-	-	-	<b>1.00 ( Ref. )</b>	-
Model 1: ‘HL’	0.007	0.114	0.459	0.008	1.08 (0.86; 1.35)	$P=0.498$
Model 2: Lsmm-Hwc	0.708	0.219	10.471		2.03 (1.32; 3.11)	<b>P&lt;0.001</b>
Model 3: Lsmm-Lwc	0.484	0.175	7.655		1.61 (1.15; 2.28)	<b>P=0.006</b>
Model 4: Hsmm-Hwc	0.319	0.112	8.119		1.37 (1.10; 1.71)	<b>P=0.004</b>
<b>‘MetS’</b>						
Model 0: ‘No-HL’ or ‘Hsmm-Lwc’	-	-	-	-	<b>1.00 ( Ref. )</b>	-
Model 1: ‘HL’	-0.142	0.126	1.256	0.210	0.86 (0.67; 1.11)	$P=0.262$
Model 2: Lsmm-Hwc	2.268	0.241	88.486		9.65 (6.02; 15.49)	<b>P&lt;0.0001</b>
Model 3: Lsmm-Lwc	0.667	0.187	12.744		1.94 (1.35; 2.80)	<b>P&lt;0.0001</b>
Model 4: Hsmm-Hwc	2.484	0.124	402.632		11.9 (9.40; 15.27)	<b>P&lt;0.0001</b>
<b>‘High CVR’</b>						
Model 0: ‘No-HL’ or ‘Hsmm-Lwc’	-	-	-	-	<b>1.00 ( Ref. )</b>	-
Model 1: ‘HL’	0.483	0.142	11.616	0.169	1.62 (1.22; 2.13)	<b>P=0.001</b>
Model 2: Lsmm-Hwc	3.072	0.330	86.611		21.59 (11.30; 41.23)	<b>P&lt;0.0001</b>
Model 3: Lsmm-Lwc	1.922	0.195	97.153		6.83 (4.66; 10.01)	<b>P&lt;0.0001</b>
Model 4: Hsmm-Hwc	2.962	0.150	391.320		19.34 (14.42; 25.94)	<b>P&lt;0.0001</b>
<b>‘NAFLD by higher GGT’</b>						
Model 0: ‘No-HL’ or ‘Hsmm-Lwc’	-	-	-	-	<b>1.00 ( Ref. )</b>	-
Model 1: ‘HL’	-0.189	0.113	2.802	0.012	0.82 (0.66; 1.03)	$P=0.094$
Model 2: Lsmm-Hwc	0.503	0.221	5.181		1.65 (1.07; 2.55)	<b>P=0.023</b>
Model 3: Lsmm-Lwc	0.000	0.183	0.000		1.00 (0.69; 1.43)	$P=0.998$
Model 4: Hsmm-Hwc	0.533	0.107	24.755		1.70 (1.38; 2.10)	<b>P&lt;0.0001</b>
<b>‘NAFLD by higher PGT’</b>						
Model 0: ‘No-HL’ or ‘Hsmm-Lwc’	-	-	-	-	<b>1.00 ( Ref. )</b>	-
Model 1: ‘HL’	-0.019	0.139	0.019	0.018	0.98 (0.74; 1.28)	$P=0.890$
Model 2: Lsmm-Hwc	0.143	0.297	0.231		1.15 (0.64; 2.06)	$P=0.631$
Model 3: Lsmm-Lwc	-0.231	0.257	0.811		0.79 (0.48; 1.31)	$P=0.368$
Model 4: Hsmm-Hwc	0.651	0.132	24.372		1.91 (1.48; 2.48)	<b>P&lt;0.0001</b>

Data are shown as mean and (95%CI) for continuous outcomes and as frequency and (% percentage) for categorical outcomes. Groups are described as: (Lsmm-Hwc) Low-skeletal muscle mass and high waist circumference phenotypical model. (Lsmm-Lwc) Low-skeletal muscle mass and low waist circumference phenotypical model. (Hsmm-Hwc) High-skeletal muscle mass and high waist circumference phenotypical model. (Hsmm-Lwc) High-skeletal muscle mass and low waist circumference phenotypical model. ( $\beta$ ) Beta. (SE) Standard error. (Wald) Wald chi-square. (OR) Odds ratios. (95%CI) 95% confidence interval. (HTN) Hypertension. (MetS) Metabolic syndrome. (CVR) Cardiovascular risk. (NAFLD) Non-alcoholic fatty liver disease. (PGT) Piruvic glutamyl transferase. (Ref.) Reference group.

## DISCUSSION

The aims of this study were; 1) to characterize the glucose and blood pressure control in adults with HLS based on four body composition phenotypes participants of the Chilean NHS16-17, and 2) to associate the HLS condition and body composition phenotypes (based on calf circumference and waist circumference) with diabetes and arterial hypertension markers. The main finding of this study was that *i*) adults with HLS who report phenotypically Lsmm-Hwc show higher SBP (+27 mmHg), DBP (+2 mmHg), Tc (+18.9), GGT (+9.7), and TSH (+11.4), and *ii*) both HLS condition and the Lsmm-Hwc phenotype are significantly associated with higher risk for the suffering of diabetes (HLS OR 1.28; Lsmm-Hwc OR 7.03), and arterial hypertension (HLS OR 1.44; Lsmm-Hwc OR 11.43) (Table 2). These results were displayed with other relevant results, including that adults with HLS who report phenotypically Lsmm-Hwc show higher Tc (+18.9), GGT (+9.7), and TSH (+11.4), and both HLS condition and Lsmm-Hwc phenotype report a high cardiovascular risk (HLS OR 1.62; Lsmm-Hwc OR 21.59) (Table 2).

Comparing No-HLS vs. HLS, HLS adults were characteristically older (57.1 vs. 46.7 y), and showed elevated FPG (102.7 vs. 99.3 mg·dL) and SBP (132 vs. 126 mmHg) vs. No-HSL peers respectively (Table 1). In this line, the Chilean adult population increase their physical inactivity prevalence ~40-45 years old ([Garrido-Méndez et al., 2019](#)), being cardiometabolic diseases being more prevalent at older ages ([Petermann, Durán, et al., 2017](#)). From here, it is not surprising to find out that HLS who are mainly older persons also show more elevated FPG and SBP. Thus, although the ageing process is not a rule suffering of both HLS conditions as well as more elevated diabetes and HTN diseases, there is an urgent need for increasing health promotion and possibilities to maintain a healthy lifestyle in older adult populations.

Physical inactivity modulates body composition, promoting SMM loss, known as sarcopenia, and increasing adiposity as an obesity environment, summarized in a body composition phenotype of low SMM, and high adiposity, as in the case of the present study is represented by the high WC (i.e., Lsmm-Hwc phenotype). By contrast, physical activity promotes the maintenance of SMM and growing and low levels of adiposity, represented phenotypically by the Hsmm-Lwc phenotype as the reference group. Previous longitudinal literature studies from (Lee et al., 2012) have revealed that after 6-years of follow-ups, adults who increased their adiposity (By body fat percentage and BMI increases) were associated with a high risk for developing HTN, metabolic syndrome and hypercholesterolemia. By contrast, those who maintained cardiorespiratory fitness were associated with lower cardiovascular risk. However, this literature study does not reported populations with disabilities as HLS. Worryingly, in the present study, participants with a Lsmm-Hwc body composition phenotype reported higher SBP (+27 mmHg) and DBP (+2 mmHg), being the risk for suffering of HTN also higher in both HLS condition (OR 1.44) and more superior in those of Lsmm-Hwc phenotype (OR 11.43) (Table 2). Thus, from each side of the condition, for example, having HLS or an unhealthy body composition phenotype, there is a higher risk of developing HTN. Therefore, there is a need for longitudinal studies, particularly on how prevalent cardiometabolic diseases are in HLS populations.

The knowledge about HL and its association with mental health still needs to be improved. Few studies have established these associations, highlighting that adults and older adults with HL tend to show conditions such as depression more than those without HL ([West, 2017](#)). Likewise, it has been postulated that depression, with other multifactorial variables, promotes limitations in daily living activities in adults with HL, which could impact a healthy lifestyle ([West, 2017](#)). This negatively impacts adherence to physical

activities, increasing sedentary behaviour. Our results did not show differences between No-HLS vs. HLS subjects in physical activity levels of PA<sub>LI</sub> and PA<sub>MI</sub>, but HLS showed to practice more PA<sub>VI</sub> (Table 1). However, these findings are not uncommon due to PA<sub>VI</sub> is usually the minor physical activity modality practised during the week and reasonably could not influence modifying body composition as SMM or WC in adults.

On the other hand, future research from a more mechanistical more than associative approach is needed to determine whether an unhealthy body composition as the Lsmm–Hwc phenotype is a risk factor for HLS, or whether the presence of HLS leads to an unhealthy body composition phenotype. Identifying the clear direction of this relationship is relevant to promote ‘health promotion’ actions to reduce the HLS or at least to promote physical activity in populations with HLS looking to avoid additional comorbidities such as diabetes and HTN that clearly would decrease their health and QoL.

Some limitations of our work included that i) we did not measure clinically HL condition by pure audiometry and measured the physical activity level by standardized questionnaires (i.e., International PA questionnaire GPAQ, and not objectively measures by accelerometry equipment), where we used the self-reported information from the NHS16-17 of these outcomes, ii) the four body composition phenotypes were modelled using secondary outcomes of SMM and adiposity as were ‘calf circumference’ and ‘waist circumference’, but both outcomes are frequently used in clinical practice, and has a strong association with sarcopenia/skeletal muscle mass and body fat, iii) although we reported a significant association between HLS and several cardiometabolic markers, the effect sizes were relatively small, and iv) our HLS sample could be not directly to be representative of some specific adult groups as those adults who work in noise environments, due to construction sites, for example, are more likely to suffer of HL (WHO, 2021a). Some strengths are that i) the NHS16-17 is a representative study of the Chilean population, ii) the present study reports information about cardiometabolic risk in adults, and iii) this information could be useful for adjusting physical activity guidelines to populations with some hearing disabilities.

Chilean adults with HLS who report phenotypically a low skeletal muscle mass and high waist circumference are characterized by registering higher SBP (+27 mmHg) and DBP (+2 mmHg). Both HLS and Lsmm–Hwc isolated conditions represent a higher risk for suffering from diabetes and HTN and increase the cardiovascular risk in adults. Future studies should explore this association in a more longitudinal than cross-sectional manner as well as exploring those mechanisms underlying these relationship.

### Conflict of interest

No conflict of interest is declared by the authors.

### Ethics Committee

The Chilean National Health Surveys were funded by the Chilean Ministry of Health and led by the Department of Public Health of the Pontificia Universidad Católica de Chile. The Chilean National Health Surveys were approved by the Ethics Research Committee of the Faculty of Medicine at the same university.

### Author contributions

Study Design, CA; Data Collection, CA, DT-R, LM; Statistical Analysis, CA; Data Interpretation, CA, DT-R, LM, CF-B; Manuscript Preparation, CA, DT-R, LM, CF-B, AR; Literature Search, All authors. All authors have read and agreed to the published version of the manuscript.

### Data Availability Statement

All data information can be found freely accessed at the Epidemiological Unit of the Chilean Health Ministry at <http://epi.minsal.cl/encuesta-ens-descargable>

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