Research article

The relationship between the postprandial lipemic response and lipid composition in persons with spinal cord injury

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Objective: To determine the influence of lipid concentration, lipid particle size, and total abdominal fat (TAF) on postprandial lipemic response (PPLr) in persons with spinal cord injury (SCI).

Methods: Thirty-five persons with SCI (17 paraplegia, 18 tetraplegia) and 18 able-bodied (AB) individuals participated. Following a 10-hour fast, blood was drawn for lipids, apolipoprotein (apo) A1 and B concentrations, and low-density (LSP) and high-density (HSP) lipoprotein particle sizes. A high-fat milkshake was consumed (~1.3 g fat/kg). Blood was drawn at 2, 4, and 6 hours to determine PPLr, (triglyceride (TG) area under the curve). TAF and visceral (VF) fat were measured by ultrasonography; total body fat (TBF) by dual-energy X-ray absorptiometry. Differences between the groups were determined by independent sample *t*-tests. Pearson correlation coefficients determined the relationship among PPLr and lipids, and TAF.

Results: There were no significant differences in fasting TG, low-density lipoprotein (LDL), apoB, TAF, or PPLr values between the groups. In SCI, PPLr significantly correlated with: apoB ($r = 0.63$, $P < 0.01$), LSP ($r = 0.57$, $P < 0.01$), and TAF ($r = 0.36$, $P < 0.01$). After controlling for age and duration of injury, PPLr significantly correlated with apoB ($r = 0.66$, $P = 0.001$), TBF ($r = 0.45$, $P = 0.03$), VF ($r = 0.66$, $P = 0.02$), and TAF ($r = 0.56$, $P = 0.007$).

Conclusions: Although concentrations of LDL cholesterol and apoB were not different between SCI and AB groups, LSP, apoB, and TAF correlated with PPLr in persons with SCI. ApoB was associated with a greater PPLr in those with motor complete SCI, after controlling for age and duration of injury.

Keywords: Postprandial lipemia, Lipid particle size, Spinal cord injury, Abdominal fat, Apolipoprotein B

Introduction

Conventional risk factors for cardiovascular disease (CVD) have been well described in the spinal cord injury (SCI) population. A high prevalence of dyslipidemia, specifically, depressed high-density lipoprotein (HDL) cholesterol and elevated triglycerides (TG), insulin resistance, and central obesity have been observed in individuals with SCI.^{1–3}

Emergent evidence suggests that the size of the low-density lipoprotein (LDL) cholesterol particle contributes to the atherogenic process. Small, dense LDL particles are able to permeate the endothelial wall, where they are oxidized and promote atherogenic plaque formation.^{4,5} Although the serum LDL concentrations have been reported to be normal in the SCI population, compared to able-bodied (AB) controls, the composition of LDL particle subfractions is not known. The combination of sedentary lifestyle, dyslipidemia, obesity, impaired glucose control, and more frequent

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urinary tract, pulmonary, and skin infections increases the inflammatory stress in persons with SCI. Orakzai *et al.*⁶ have reported that persons with SCI are more likely to have premature accumulation of coronary artery calcium plaque, which is indicative of CVD.

It has been well established that an accumulation of adipose tissue in the abdominal region can predispose an individual to CVD both in the AB and SCI populations. Abdominal obesity has been shown to be a stronger predictor of CVD, compared to total body fat (TBF) measures.^{7,8} Previous investigations have shown that individuals with SCI are more susceptible to elevated levels of abdominal fat due to sedentary lifestyle, alterations in metabolic rate and autonomic function, higher prevalence of dyslipidemia and impaired glucose control, duration and level of injury.^{3,9,10} Abdominal adiposity can be challenging to measure in the SCI population via traditional techniques such as waist circumference (WC), skinfolds, and bioelectrical impedance analysis. More recently, computed tomography, magnetic resonance imaging, dual-energy X-ray absorptiometry (DXA), and ultrasound have been able to provide more accurate quantification of abdominal adipose tissue.^{9–11}

Postprandial lipemia, the elevated fat levels in the blood after a meal, has been demonstrated to be associated with the progression of CVD. Zilversmit⁴ suggested that postprandial lipemia may, in part, be the result of inflammatory processes. Individuals with SCI are often in a state of systemic inflammation due to the variety of conditions related to SCI, including pressure ulcers, chronic urinary tract infections, atelectasis, and respiratory tract infections. Thus, individuals with SCI who are predisposed to ongoing inflammatory processes may have decreased rates of postprandial lipid clearance, which may contribute to their heightened risk of CVD. The purpose of this study was to determine the relationship among postprandial lipemic response (PPLr) after an oral fat load and lipid composition and abdominal fat in persons with SCI.

Methods

Subjects

Eighteen healthy AB subjects (17 males, 1 female) and 35 subjects with chronic, stable, SCI (32 males, 3 females) between the ages of 20 and 65 years of age were recruited for study participation. Subjects with SCI were matched by age (± 10 years) to an AB subject with similar demographic characteristics. Those with SCI were included if they had a chronic injury (≥ 1 year), regardless of completeness or

mechanism of injury, and if they were clinically stable. Seventeen subjects were diagnosed with paraplegia, and 18 subjects with tetraplegia. The duration of injury (DOI) in the SCI group ranged from 1 to 45 years. Of the 35 subjects with SCI, 21 subjects were classified according to the American Spinal Injury Association (ASIA) Impairment Scale (AIS) as A, 4 subjects as AIS B, 8 subjects as AIS C, and 2 subjects as AIS D. The two subjects classified as AIS D used manual wheelchairs more than 80% of the time, and each was not able to stand and/or ambulate more than a few steps without assistance. Individuals were excluded from participation if they had an acute illness, a clinical diagnosis of heart, pulmonary, endocrine disease (including diabetes), were currently being treated for dyslipidemia, had a dairy and/or chocolate allergy, or if they were SCI and could ambulate.

Procedures

Subjects underwent two non-consecutive days of testing at the Department of Veterans Affairs Rehabilitation Research and Development National Center of Excellence for the Medical Consequences of SCI at the James J. Peters VA Medical Center (JJPVAMC) or Kessler Institute for Rehabilitation (KIR). Following informed consent in accordance with the policies of the Institutional Review Boards of the JJPVAMC and KIR, subjects underwent a series of anthropometric measurements, venipuncture with blood collection, and a medical history, including obtaining conventional risk factors for CVD by interview. Investigators were not blinded to subject group assignment.

Blood lipids

Fasting blood samples were drawn after at least a 10-hour fast for measurement of fasting serum lipids and plasma insulin. Serum total cholesterol (TC), TG, and HDL cholesterol levels were determined using an ADVIA 1650 automatic chemistry analyzer using the procedures recommended by the manufacturer (Bayer Diagnostics, Newbury, UK); LDL levels were estimated using the Friedewald equation.¹² Determination of fasting serum LDL cholesterol particle sizes were measured via nuclear magnetic resonance spectroscopy (NMR, LipoScience Inc., Raleigh, NC, USA). Each lipoprotein subclass emits different proton NMR amplitude and shape of the waveform, which are used to determine the concentration of each lipoprotein subclass. Fasting serum apolipoprotein A1 (apoA1) and apolipoprotein B (apoB) concentrations were measured by a commercial laboratory (Quest Diagnostics, Teterboro, NJ, USA).

Anthropometric and body composition measurements

Subjects wore a lightweight shirt and pants or shorts without metal parts during the body composition and anthropometric measurements. All extraneous clothing, shoes, and metal objects (watches, rings, necklaces, etc.) were removed prior to testing. Body composition, height, weight, and waist and hip circumferences were measured.

DXA (LUNAR Prodigy and iDXA, GE Lunar, Madison, WI, USA) imaging was performed to determine body composition, height, and weight. Identical DXA scanners were located at both study locations. During the course of the study, DXA scanner models were upgraded from Prodigy to iDXA. Upgrades from Prodigy to iDXA include the ability to scan heavier (up to 450 pounds iDXA vs. 300 pounds Prodigy) and larger (up to 66 cm wide $\times$ 198 cm long iDXA vs. 60 cm wide $\times$ 197.5 cm long Prodigy) subjects. The number of detectors increased from 16 in the Prodigy to 64 detectors in the iDXA, permitting greater image resolution.¹³ To account for differences in the estimation of measurement, values for total body bone mineral content, lean, and fat mass collected on the Prodigy were translated to iDXA values via regression equations described by Hull *et al.*¹⁴ Thirty-eight subjects were scanned by the Prodigy DXA, and 15 subjects were scanned by the iDXA.

Subjects were positioned on the imaging table for a total body scan. To accommodate individuals with SCI who also had contractures and/or muscular pain, particular effort was made to position each subject as comfortably, yet as precisely, as possible. Total body scan time lasted 7–12 minutes (thin and standard thickness scans vs. thick scans). Regions of interest were adjusted for each image to determine percent TBF and images were analyzed by one investigator. Body weight was recorded as the total body mass from the DXA scan. Height was determined from the scan using electronic calipers to measure the length from the top of the skull to the calcaneus. Body mass index (BMI; kg/m²) was calculated from the weight and length measures obtained from the DXA scan using the standard formula.¹⁵ TBF was calculated as follows: $(\text{TBF (g)}/\text{total body weight (g)}) \times 100$. Daily quality assurance testing was performed on each DXA scanner; the coefficient of variation was <1% at both study locations for 150 phantom scans, in which the reference range was 30% fat.

While subjects were in the supine position on the DXA table, a flexible anthropometric measuring tape was wrapped around the waist at the level of the umbilicus to measure WC. Hip circumference was measured

at the level of the iliac crest. Three measurements were taken at each site and the average of the values was recorded. The ratio of the waist-to-hip (WH) circumferences was calculated.

Abdominal ultrasound

Subjects were positioned supine on a table for ~20 minutes while the ultrasound examination was performed by a trained sonographer. All scans were obtained using the GE Logiq Book XP (GE Healthcare, Buckinghamshire, UK) using a 3C-RS 2–5 MHz curvilinear transducer (GE Healthcare) to measure subcutaneous and visceral adipose tissues.^{16,17} Identical ultrasound units were located at both study sites. Subcutaneous fat (SF) was measured with the transducer set at 5 MHz positioned transversely with minimal pressure applied so as not to compress the adipose tissue below. The area 1 cm above the umbilicus along the xiph umbilical line was measured upon exhalation as the distance between the skin–fat interface and the outer edge of the linea alba. Visceral fat (VF) was measured in transverse as the distance between the linea alba and the anterior aorta, 1 cm above the umbilicus along the xiph umbilical line at the end of exhalation. Three measurements were recorded and averaged for the determination of SF and VF thicknesses. Total abdominal fat (TAF) was calculated as the sum of SF and VF.¹⁶

Fat meal challenge

On Day 2, the subjects underwent an oral fat challenge after at least a 10-hour fast. A baseline blood sample was obtained for measurement of fasting serum TGs. Subjects then drank a chocolate milkshake consisting of premium ice cream and heavy whipping cream. This milkshake delivered ~1.3 g fat, 1.2 g carbohydrate, and 0.2 g of protein per kilogram of body mass. To accommodate for the differences in body size, the fat concentration of the milkshake administered, but not its volume, varied with the weight of the individual. The test drink was administered over a 20-minute period, and subjects were instructed to take nothing by mouth except for water during the 6-hour period of testing. Blood was drawn for measurement of serum TGs at 2, 4, and 6 hours after administration of the fat load. Subjects were required to abstain from physical work or exercise during the postprandial period. The PPLr was measured as the area under the curve (AUC) for TG values at each timepoint from baseline to 6 hours, and AUC was calculated using the trapezoid method.

Statistical analysis

The data were analyzed using IBM SPSS Version 19 (Armonk, NY, USA). An *a priori* alpha level was set

at $P \leq 0.05$. Demographic and descriptive data are reported as mean $\pm$ standard deviation. Independent samples *t*-tests were employed to determine differences between the groups (AB vs. SCI, PARA vs. TETRA, and complete SCI vs. incomplete SCI) for each of the independent variables (e.g. age, weight, height, BMI, WC, WH, fasting serum TC, TG, HDL cholesterol, LDL cholesterol, lipid particle sizes, apoA1, apoB, TBF, VF, TAF, and TG AUC). Pearson correlation coefficients determined the relationship among PPLr AUC and lipids and TAF. Partial correlations were used to determine the relationship among PPLr AUC and body fat measures while controlling for age and DOI in motor complete SCI. To predict PPLr, while controlling for age and DOI in motor complete SCI, multiple stepwise regression was utilized.

Results

The ages of subjects ranged from 25 to 61 years (Table 1). Age, height, and weight were not statistically different between the groups. DOI was significantly greater in persons with tetraplegia compared to those with paraplegia (23 ± 12 vs. 14 ± 11 years, respectively; $P = 0.04$).

Fasting lipid concentrations

No significant differences were noted in fasting TC and LDL cholesterol concentrations between the groups (Table 2). Fasting TGs were significantly greater in the SCI group compared to the AB group (146.2 ± 87.1 vs. 102.0 ± 65.6 mg/dl, respectively, $P = 0.05$). The group with SCI had significantly lower HDL cholesterol concentrations than that of the AB group ($P < 0.05$). The apoA1 concentration was significantly lower in the SCI group than in the AB group ($P < 0.01$). Subjects with motor complete SCI, in particular, were shown to have lower apoA1 compared to AB subjects (131.5 ± 20.6 vs. 145.9 ± 23.8 mg/dl respectively; $P = 0.04$.) No differences were observed in apoB, large (LLP) or small LDL particle (LSP) concentrations between the groups. Large (HLP) and small HDL (HSP) particle concentrations trended to be higher in the AB group than the SCI group (Table 2, HLP: 7.19 ± 3.89 vs. 5.21 ± 2.54 $\mu\text{mol/l}$, respectively, $P = 0.06$; HSP: 22.08 ± 4.99 vs. 19.08 ± 6.60 $\mu\text{mol/l}$, respectively, $P = 0.07$).

Anthropometrics and measures of body composition

No significant differences were noted in WH, VF, or TAF between the groups. Subjects in the SCI group had significantly greater TBF and WC compared to

subjects in the AB group (Table 3, TBF: 34.5 ± 7.5 vs. $26.2 \pm 7.4\%$, $P < 0.01$, WC: 99.7 ± 14.9 vs. 91.5 ± 12.4 cm, $P < 0.05$, respectively). No significant differences were observed in WC and VF between motor complete and incomplete SCI. TBF measured by DXA was significantly greater in subjects with motor complete SCI compared to motor incomplete SCI (36.7 ± 5.5 vs. $30.9 \pm 10.5\%$, respectively; $P = 0.04$) and AB subjects ($26.7 \pm 7.6\%$, $P < 0.001$).

TG responses

There were no differences between the groups in the responses (Fig. 1) at 2 hours (AB vs. SCI: 199.4 ± 105.6 mg/dL vs. 146.2 ± 87.1 mg/dL, $P = 0.63$), 4 hours (AB vs. SCI: 290.9 ± 218.1 vs. 294.4 ± 138.6 mg/dL, $P = 0.92$), or 6 hours (AB vs. SCI: 277.9 ± 211.1 vs. SCI: 283.1 ± 173.9 mg/dL, $P = 0.99$). Subjects with paraplegia had an elevated TG concentration at the 2-hour timepoint compared to those with tetraplegia (261.3 ± 118.4 vs. 180.3 ± 62.6 mg/dL respectively; $P = 0.02$), as well as subjects with motor complete SCI compared to motor incomplete SCI (241.6 ± 108.5 vs. 164.9 ± 51.2 mg/dL, respectively; $P = 0.04$). Both groups reached peak postprandial TG concentration at the 4 hours after the oral fat load (Fig. 1). There were no significant differences in PPLr between the groups (AB vs. SCI: 1271 ± 756 vs. 1449 ± 8691 mg/dl, $P = 0.41$). However, subjects with motor complete SCI did trend to have a more prolonged TG clearance rate compared to motor incomplete SCI (1590 ± 752 vs. 1097 ± 318 mg/dl respectively; $P = 0.06$).

In the individuals with SCI (both motor complete and incomplete), PPLr significantly correlated with apoB ($r = 0.625$, $P < 0.001$, Fig. 2), LSP ($r = 0.572$, $P < 0.001$, Fig. 3), TBF ($r = 0.355$, $P < 0.05$), and TAF ($r = 0.358$, $P < 0.01$). In the AB controls, PPLr significantly correlated with LSP ($r = 0.632$, $P < 0.01$), WC ($r = 0.47$, $P < 0.05$), TAF ($r = 0.624$, $P < 0.01$), and inversely with HLP ($r = -0.536$, $P < 0.01$). ApoB concentrations correlated significantly with concentrations of both large and small LDL particle size in the SCI group (LLP: $r = 0.43$, $P < 0.01$ and LSP: $r = 0.616$, $P < 0.01$), whereas in the AB group, apoB concentration only correlated with LSP concentration ($r = 0.745$, $P < 0.01$). When controlling for age and DOI, in subjects with SCI (both motor complete and incomplete), apoB ($r = 0.62$, $P < 0.001$), and TAF ($r = 0.40$, $P = 0.02$) correlated with PPLr. When controlling for age and DOI in participants with motor complete SCI, apoB ($r = 0.66$, $P = 0.001$), TBF% ($r = 0.45$, $P = 0.03$), VF ($r = 0.47$, $P = 0.02$), and TAF ($r =$

Table 1 Subject demographics

	AB (<i>n</i> = 18) (range)	SCI (<i>n</i> = 35) (range)
Age (years)	42 ± 11 (27–60)	44 ± 10 (25–61)
Height (m)	1.75 ± 0.09 (1.62–1.91)	1.78 ± 0.07 (1.60–1.91)
Weight (kg)	83.7 ± 15.8 (61.4–118.0)	82.4 ± 17.6 (51.3–120.4)
BMI (kg/m ²)	27.4 ± 4.0 (22.0–35.2)	26.4 ± 5.4 (17.1–41.7)
Male/female	17/1	32/3
DOI (years)		19 ± 12 (1–45)
Complete lesion (<i>n</i>)		25
Incomplete lesion (<i>n</i>)		10
Paraplegia/tetraplegia		17/18

AB, able-bodied; SCI, spinal cord injury; BMI, body mass index; DOI, duration of injury.

0.56, *P* = 0.007) significantly correlated with PPLr. In subjects with motor complete SCI, the strongest predictors of PPLr were apoB and TAF ($R^2 = 0.603$, *P* = 0.001). The model equation is depicted: postprandial TG response = $-7.268 + (0.100 \times \text{age}) - (0.331 \times \text{DOI}) + (0.203 \times \text{apoB}) + (1.406 \times \text{TAF})$.

Discussion

The prevalence of dyslipidemia in persons with SCI has been thoroughly described.^{3,18–21} However, additional factors related to the PPLr, other lipid particles, and body composition, which have been postulated to contribute to the increased risk and prevalence of coronary heart disease in the general population, have been previously investigated in persons with SCI.^{3,10,22}

Depressed serum HDL cholesterol and elevated serum TGs have been identified as the lipid risk factors for CVD in persons with SCI, with serum LDL cholesterol being relatively normal, compared to age- and sex-matched AB control subjects.^{1,3,18} Despite the presumption that CVD occurs prematurely,

and is more prevalent in those with SCI than in matched AB controls, the treatment strategies for reducing the risk of CVD associated with dyslipidemia in persons with SCI are similar to those in the AB population. In the AB population, reports have suggested that the composition of lipid particle sizes, regardless of the total serum LDL concentration, may be a contributing factor to the progression of CVD.^{23,24} Small, dense LDL particles are capable of permeating the endothelial wall and become oxidized, stimulating atherosclerotic plaque formation.²³ To date, no reports have addressed LDL particle subfraction concentrations in persons with SCI. Somewhat surprisingly, it appears that individuals with SCI were not found to have a greater concentration of small, atherosclerotic LDL particles to that found in AB controls.

As described in this report and by other investigators, individuals with SCI have significantly greater TBF compared to AB individuals. Paralysis of major muscle groups and the sudden and dramatic decrease in the level of physical activity undoubtedly contributes

Table 2 Fasting lipid measurements

	AB (<i>n</i> = 18)	SCI (<i>n</i> = 35)
TC (mg/dl)	187.7 ± 36.5	179.8 ± 35.4
TG (mg/dl)	102.6 ± 65.6	146.2 ± 87.1*
LDL (mg/dl)	115.7 ± 33.6	109.1 ± 27.2
HDL (mg/dl)	51.4 ± 13.4	43.1 ± 9.2*
ApoA1 (g/l)	1.46 ± 0.24	1.27 ± 0.22*
ApoB (g/l)	0.86 ± 0.24	0.88 ± 0.21
ApoB100 (g/l)	0.70 ± 0.50	0.84 ± 0.39
LLP (nmol/l)	409.4 ± 181.1	409.3 ± 170.0
LSP (nmol/l)	748.6 ± 384.0	842.5 ± 369.1
HLP (nmol/l)	7.2 ± 3.9	5.2 ± 2.5
HSP (nmol/l)	22.1 ± 5.0	19.1 ± 6.6

AB, able-bodied; SCI, spinal cord injury; TC, total cholesterol concentration; TG, triglyceride concentration; LDL, low-density lipoprotein concentration; HDL, high-density lipoprotein concentration; ApoA1, apolipoprotein A1; ApoB, apolipoprotein B; ApoB100, apolipoprotein B100; Lp(a), lipoprotein (a); LLP, large LDL particle concentration; LSP, small LDL particle concentration; HLP, large HDL particle concentration; HSP, small HDL particle concentration.

**P* < 0.05.

Table 3 Anthropometrics and measures of body composition

	AB (n = 18)	SCI (n = 35)
TBF (%)	26.2 ± 7.4	34.5 ± 7.5*
WC (cm)	91.5 ± 12.4	99.7 ± 14.9*
WH	0.92 ± 0.07	0.96 ± 0.10
VF (cm)	5.38 ± 2.01	5.09 ± 2.44
TAF (cm)	7.27 ± 2.46	6.49 ± 2.60

AB, able-bodied; SCI, spinal cord injury; TBF, total body fat by DXA; WC, waist circumference; WH, waist-to-hip ratio; VF, visceral fat by ultrasound; TAF, total abdominal fat by ultrasound.

*P < 0.05.

to the accumulation of TBF. Other factors that influence negative body composition changes in this population include decreased metabolic rate, disruption of the autonomic nervous system, and an imbalance between energy intake and expenditure. Our group has recently found that there is an approximate 10 kg increase in total body adiposity, with a gain in percent adiposity from 24 to 35%, over the initial 24 months after acute SCI (unpublished data). Greater WCs in persons with SCI suggest greater visceral adiposity compared to AB individuals. However, we observed quite comparable amounts of VF and TAF measured by ultrasound. Greater WC in those with SCI compared to AB subjects may be due to altered fat distribution in persons with SCI across the abdominal region, subtle positioning differences between the groups at the time of measurement (all subjects were measured while supine), reduction of abdominal wall musculature, and possibly, greater bowel distention secondary to increased constipation in subjects with SCI.²⁵ Ultrasound is not as sensitive in the measurement of abdominal fat, because abdominal fat may not be deposited at the measurement site, whereas it may collect laterally or in the posterior areas of the trunk. Our previous work has found that

men with paraplegia had greater subcutaneous abdominal fat compared to age-matched AB subjects.^{10,25} The use of abdominal ultrasonography for the measurement of VF has been shown to be an accurate surrogate measure compared to computed tomography in AB subjects, but not in the SCI population.^{16,17} Our findings suggest that it may be possible that after SCI, fat is distributed differently across the abdominal region than in the AB individuals. Further investigations with MRI and CT to determine the differences in distribution of fat in the various abdominal compartments should be considered.

Currently, there are only a few reports describing the PPLr after a liquid oral fat load in persons with SCI.^{10,22} Nash *et al.*²² reported that the PPLr to an oral fat load, which was identical in composition as the fat load administered in our study, was “exaggerated” in 3 active individuals with paraplegia, compared to that observed in 21 AB controls. The findings from our previous report are in agreement with those from this current study, in which individuals with paraplegia disposed of an oral fat load similarly to that of AB controls, without

Figure 1 TG concentrations at baseline, 2, 4, and 6 hours after administration of a standard fat meal challenge.

Figure 2 Relationship among PPLr as measured by the TG AUC and fasting serum apoB concentration ($r = 0.625$, $P < 0.001$) in the SCI group only.

Figure 3 Relationship among PPLr as measured by the TG AUC and fasting total small LDL particle concentration ($r = 0.355$; $P < 0.05$) in the SCI group only.

observing an “exaggerated” response in those with SCI.¹⁰ In prior reports that have studied the PPLr in persons with SCI, the cohorts investigated have been relatively small, and may not be representative of the SCI population in general.²²

A relationship was observed between apoB concentrations and LDL particle size to that of PPL response in the SCI group only. Individuals with SCI who had higher fasting serum concentrations of apoB took a longer time to clear lipids from the circulation (greater PPL time, greater AUC), and they also had greater fasting serum concentrations of large and small LDL particles. In AB subjects, the fasting serum concentration of apoB was associated with fasting small LDL particle concentration. Although our study described the relationship between fasting apoB concentrations and PPL responses, Masuda *et al.*²⁶ reported that after an oral fat load, fasting apoB-48 concentrations correlated with TG AUC after a high-fat meal; a correlation was also observed for fasting and stimulated levels of apoB-48. These findings suggest that a high-fat meal results in higher postprandial TG concentrations because of elevated apoB-48 remnant production from the small intestine, with little increase in serum LDL-c hepatic production of apolipoprotein B100.^{27,28} After administration of a fat meal consisting of 33.3% fat, Sato *et al.*²⁹ reported a similar direct relationship between fasting TG and apoB-48 concentrations and postprandial TG concentration. Thus, it may be worthwhile to characterize baseline levels of apoB-48 to determine which patients are at an increased risk for an elevated PPLr; this approach may also be performed, *albeit* not as easily, for other remnant lipid particles while fasting.²⁹ Further investigation is needed to determine

the rate of lipid metabolism in persons with SCI, and to determine the proper types of fats that should be consumed, that would not pose an adverse influence on the CVD risk profile.

Limitations

The use of two different DXA scanner models was not ideal to determine body composition between the groups in this investigation. Although it was not possible to maintain the DXA Prodigy scanner for the duration of our study, a published report does provide conversion factors between the Prodigy and the iDXA for soft tissue mass.¹⁴ Accurate measurement of height and WC is rather challenging in the SCI population. Standard protocols require subjects to be in the standing position. Because standing can be difficult to achieve in persons with SCI, these measurements are generally obtained in either the supine or seated positions. Seated WC measures could potentially overestimate abdominal fat due to the fat depot residing along the WC, and an inability for the subject to stretch the torso. By placing the subject in the supine position, the torso can be stretched, simulating a standing position, which may provide a better approximation of WC. Although not ideal, determining length while supine is most likely more accurate than relying on the recording of subjectively reported height. Contractures of the lower extremity and scoliosis of the spine are prevalent in the SCI population which effect height; however, attempting to straighten the legs and spine within comfortable limits and measuring the body supine is an acceptable alternative.

Comparisons among studies administering oral fat loads can be challenging due to the lack of standardization of meal content (i.e. liquid vs. solid) and composition (i.e. grams of fat, carbohydrate, and protein).³⁰ In addition to varying compositions, duration of observation and intervals of blood collection are not consistent across reports. Of note, a majority of the studies observe the TG response for 6–8 hours after meal administration; however, the postprandial TGs may take ~24 hours to completely clear from the circulation. Although individuals with SCI have similar TG clearance rates to AB controls between baseline and 6 hours postprandially, because baseline levels of TGs trended to be higher in those with SCI, it would be interesting to determine the clearance rate of TGs after the oral fat load at later timepoints, such as those between 12 and 18 hours postprandially. *Albeit* purely conjecture by the authors, if individuals with SCI were shown to have a greater delayed total TG clearance time at later timepoints compared to AB persons, a delayed

clearance rate may yet be found to contribute to the increased risk of CVD in persons with SCI.

There are only a few studies that describe the postprandial response to an oral fat load in subjects of varying levels and completeness of SCI.^{10,22} Thus, this investigation should serve as a starting point that will hopefully stimulate further work in larger study cohorts in an effort to determine if subgroups of persons with SCI have abnormal postprandial lipid responses compared to that of matched AB subjects, and if so, the mechanism for this finding.

Conclusion

A significant difference was not observed between the SCI and AB groups for PPLr. However, persons with motor complete SCI did trend to have decreased postprandial lipid clearance rates compared to motor incomplete SCI. Further investigation with a larger sample is necessary to determine the factors of lipid metabolic rate. In addition, when controlling for age and DOI, fasting apoB and VF measured by ultrasound strongly predict PPLr. Individuals with SCI have been reported to be predisposed to medical consequences that may contribute to a heightened progression of CVD and the risk for a morbid cardiac event, possibly including their PPLr and VF mass. Future investigations should further examine the influence of decreased metabolic rate, autonomic dysfunction, and abdominal adiposity PPLrs in persons with SCI.

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Disclaimer statements

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Conflicts of interest None.

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