Factor Analysis of the Metabolic Syndrome in Spinal Cord–Injured Men

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Disturbances of carbohydrate and lipid metabolism in men with spinal cord injury are common, but poorly defined. Clustering of recognized risk factors for obesity and disorders of carbohydrate and lipid metabolism are characteristic of the metabolic syndrome. The purpose of this study was to investigate the presence of metabolic syndrome using modifications of the World Health Organization (WHO) definition and including total physical activity levels (minutes/week), in a group of active males with spinal cord injury who were carefully matched for age, height, and weight with active able-bodied males. Factor analysis is used widely to explore factors of the metabolic syndrome. This technique was used in this study of 20 spinal cord–injured (SCI) men and 20 able-bodied controls, matched for age, height, and weight. Three-factor models, each reflecting a different aspect of the metabolic syndrome, were identified for both study groups. The average communality score for the SCI group was 0.8 and 0.7 for the control group. For the SCI group, factor 1 reflected an interaction between adiposity measures, physical activity, and postload insulin and glucose, factor 2 was reflective of dyslipidemia, while factor 3 revealed an interaction between fasting levels of insulin and glucose. In the control group, factor 1 reflected an association between the adiposity measures and physical activity, factor 2 was reflective of postload glycemic control, with factor 3 reflecting an interaction between fasting insulin and dyslipidemia. By summation of the total variance of each factor, the 3-factor models explained 80% and 69% of the variance in the original 9 variables examined in the SCI and control groups, respectively. In summary, while the WHO definition for the metabolic syndrome appears suitable for use in identifying the incidence of this syndrome in SCI men, some modification of anthropometric and lipid measures may be required.

SPINAL CORD INJURY (SCI) is strongly associated with an increased incidence of non–insulin-dependent diabetes mellitus (NIDDM) and coronary heart disease (CHD) in younger SCI age groups than in comparable able-bodied individuals.[1-3] These metabolic changes are due, in part, to the major body composition changes observed in SCI persons and may ultimately lead to the development of the metabolic syndrome (insulin resistance syndrome) and diabetes and CHD.[1,3-5] The evidence for the development of metabolic syndrome in SCI is strongly supported by the identification of specific risk factors in this population, eg, hyperinsulinemia, insulin resistance, glucose intolerance, dyslipidemia, and obesity.[1,3] Within the SCI population however, there is no conclusive evidence to demonstrate specific metabolic risks that predispose this population to the metabolic syndrome. The purpose of this study was to investigate the relationship of metabolic variables associated with the onset of the metabolic syndrome in a SCI population using factor analysis. The World Health Organization (WHO) definition of metabolic syndrome was used with modifications of the anthropometric and lipid variables for the SCI group and total physical activity levels (minutes/week) were included. Factor analysis allows inter-related variables to be reduced to coherent subsets, which are relatively independent of each other.[6] These represent distinct associations that account for the major proportion of the variance within the original variables.[6] This has permitted us to identify risk factors characteristic of the metabolic syndrome in a defined SCI and control population, which we present in this report.

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MATERIALS AND METHODS

Subjects

Twenty men (aged between 16 and 52 years) who had sustained a traumatic injury to the spinal cord participated in this study, which had ethical approval from the Ethics Committees of the Health Funding Authority, Otago (Protocol number: 97/10/084) and Canterbury (Protocol number: 99/05/067). Written, informed consent was obtained from all participants. All SCI males had sustained their injury for longer than 1 year. Within the SCI group, there were 11 tetraplegic participants (lesion levels, C4 to C7) and 9 paraplegic participants (lesion levels, T5 to L5). Their mean duration of injury was 10.3 ± 1.8 years. Each SCI participant was age-, height-, and weight-matched with an able-bodied control male. None of the SCI or able-bodied participants was diabetic or reported using medications known to affect carbohydrate or lipid metabolism.

Data Collection

Anthropometric measures of adiposity commonly used in the able-bodied population are unsuitable for SCI. Body mass index (BMI), for example, has been shown to significantly underestimate adiposity in SCI men.[4] Therefore, a more precise measure of adiposity using dual energy x-ray absorptiometry (DXA, LUNAR DPX-L; Lunar, Madison, WI) was used in this study.[5,7] Total body and regional body fat mass (FM) were measured in all participants, with total body fat percentage and trunk FM (a measure of central obesity) used as indicators of adiposity in both the SCI and able-bodied groups for the factor analysis.

Plasma glucose and insulin were measured in all participants following an overnight fast and again 2 hours after the ingestion of a 75-g oral glucose load (POLYCOSE; Ross Products Division, Abbott Laboratories, Columbus, OH). Glucose was analyzed by the hexokinase method on a Cobas Mira Plus (Roche Diagnostics, Indianapolis, IN) auto-analyzer using Roche reagents. Insulin was analyzed by radioimmunoassay (RIA), using the Coat-A-Count assay (Diagnostic Products,

Los Angeles, CA), on a Cobas Mira Plus (Roche Diagnostics) autoanalyzer.

Fasting blood samples for lipid analyses were taken in conjunction with the oral glucose tolerance test (OGTT). Total cholesterol (TC) was determined using the enzymatic cholesterol oxidase method (Abbott reagent), on an Aeroset analyzer (Abbott Laboratories, Abbott Park, IL). High-density lipoprotein (HDL) cholesterol was analyzed using the direct assay method on the Aeroset system (Abbott Laboratories Diagnostic Division) and the ratio of TC to HDL was calculated. In accordance with published studies,[8-11] fasting and 2-hour glucose and insulin values were used as markers of hyperinsulinemia, insulin resistance, and glucose intolerance, while HDL and the atherogenic ratio of TC/HDL were used as markers of dyslipidemia.

Metabolic syndrome in men is defined by the WHO as insulin resistance in the top 25% of the population as measured by the euglycemic/hyperinsulinemic clamp or the presence of impaired glucose tolerance (IGT) or type 2 diabetes and the presence of at least 2 of the following: WHR > 0.90 or BMI ≥ 30 kg/m^2; triacylglycerol (TAG) $\geq$ 1.7 mmol/L or HDL < 0.9 mmol/L; blood pressure (BP) $\geq$ 160/90 mm Hg; or microalbuminuria.[12] These criteria were used to identify the presence of the metabolic syndrome in the control group. While the WHO guidelines are suitable for use in the able-bodied population, some criteria have little applicability to the SCI population. As alluded to earlier, healthy BMI values have been shown to underestimate adiposity in the SCI population.[4] Furthermore, blood pressure measurements have little diagnostic value for predicting metabolic syndrome in the SCI population, as hypertension is not recognized as a clinical problem in SCI persons,[13,14] and BP variability is increased.[15] To identify the presence of the metabolic syndrome in individual SCI participants, we used body fat percentage values from the total body DXA scan of $\geq$25% as a marker of obesity, 2-hour insulin of $>$60 μIU/mL for hyperinsulinemia, and HDL < 1.04 mmol/L for dyslipidemia. As little consensus has been found for TAG levels in SCI persons,[16,17] we have used the HDL value from the Adult Treatment Panel III recommendations for identification of the metabolic syndrome.[18] IGT was defined according to WHO recommendations as fasting glucose < 7.8 mmol/L and a 2-hour glucose value > 11.0 mmol/L following a 75-g OGTT and diabetes as fasting glucose ≥ 7.8 mmol/L or 2-hour glucose ≥ 11.0 mmol/L.[12]

Due to the positive contribution activity can have upon the development and management of diabetes and CHD,[19] activity (total min $\cdot$ wk^{-1}) was also included as a variable in the factor analyses for both groups. Current regular weekly physical activity levels were gathered via questionnaire from both the SCI and control groups. SCI were asked to report only physical activity undertaken for purposes other than rehabilitation. Activity was classified as "strenuous" (vigorous exercise that increased heart rate significantly), "moderate" (exercise that could be continued for long periods of time), or "mild" (exercise that required minimal effort). Participants were asked to report the number of times they participated in exercise and the average duration of each session at each of the 3 intensity levels. The amount of exercise (minutes per week) for each participant was taken as the product of the number of exercise sessions reported at each intensity level and the duration of each session. Total minutes of exercise for the strenuous, moderate, and mild exercise intensities were then summed.

Statistical Analysis

All data that violated normal distribution were log transformed (base 10) for all further analyses. This specifically related to HDL, fasting insulin, postload glucose, and insulin values. Independent *t* tests were performed to determine differences between the SCI and control groups for body composition and blood analyses. Pearson correlation coefficients were determined to investigate the relationships among the chosen variables. Correlations were accepted if the significance level was $P < .05$. SPSS statistical software (SPSS for Windows, v11.0, Chicago, IL) was used for all analyses.

Table 1. Characteristics (mean ± SEM) of the Study Groups

Variable	SCI	Control
Age (yr)	33 ± 2	33 ± 2
Body fat (%)	27.5 ± 2.3*	17.8 ± 1.5
Trunk fat mass (kg)	11.2 ± 1.3*	7.1 ± 0.8
Activity (min $\cdot$ wk^{-1})	376 ± 59	312 ± 46
Fasting glucose (mmol $\cdot$ L^{-1})	5.20 ± 0.10	5.46 ± 0.10
Postload glucose (mmol $\cdot$ L^{-1})	7.20 ± 0.63*	4.48 ± 0.27
Fasting insulin (μU $\cdot$ mL^{-1})	8.09 ± 0.96	7.23 ± 0.70
Postload insulin (μU $\cdot$ mL^{-1})	64.03 ± 11.87*	17.76 ± 1.47
TC (mmol $\cdot$ L^{-1})	4.56 ± 0.16	4.15 ± 0.18
HDL (mmol $\cdot$ L^{-1})	1.07 ± 0.05*	1.27 ± 0.05
TC/HDL	4.44 ± 0.24*	3.33 ± 0.16

Abbreviations: SEM, standard error of the mean; SCI, spinal cord injured; HDL, high-density lipoprotein cholesterol; TC, total cholesterol.

*Significantly different from controls at $P < .05$.

Factor Analysis

Factor analysis was undertaken using SPSS statistical software. The principal component method of factor extraction was used. This method extracts the maximum variance of each component from the data set.[6] The components are then ordered, with the first component accounting for the maximum variance in the data and the last component associated with the least variance.[6] By default, SPSS retains only those components with an Eigenvalue greater than 1.0. Orthogonal (varimax) factor rotation was undertaken to facilitate the interpretation of the components identified in the principal components analysis. With varimax rotation, the variance of loading within factors, across variables is maximized, thus the interpretation of each factor is made easier as the variables that correlate with that factor are clearly identified.[6] Variables with a factor loading ≥ 0.4 were considered to be significant constituents of that factor.

RESULTS

Table 1 shows that the SCI group had higher total body fat percentage, greater trunk fat, higher postload glucose and insulin values, lower HDL, and higher TC/HDL ratios that the controls. No significant difference ($P > .05$) was observed for age, activity, TC, fasting glucose, and fasting insulin between the SCI and control groups (Table 1). Physical activity results revealed both groups comprised highly physically active individuals; the New Zealand average for physical activity is 2.5 hours $\cdot$ wk^{-1} (150 min $\cdot$ wk^{-1}).[20]

Pearson correlation coefficients are shown in Table 2. Trunk fat and fat percent were significantly correlated in both groups. Positive, significant associations between postload glucose and both fat percent and trunk fat were also found in both the SCI and control groups. Further significant positive correlations were observed in the SCI group for postload insulin and both measures of adiposity, TC/HDL, and postload glucose. Inverse associations were observed for activity with the adiposity measures, TC/HDL, and postload glucose; and for HDL with TC/HDL, and postload insulin in the SCI group. Fasting values of insulin and glucose were also inversely correlated with each other in the SCI sample. In the control group, inverse associ-

Table 2. Pearson Correlation Coefficients for the Relationships Between Factor Analysis Variables for the SCI and Control Groups

Variable	Fat %	TF	Activity	TC/HDL	Log HDL	FPG	Log G_{120}	Log FPI	Log L_{120}
Fat (%)		0.91†	−0.67†	0.31	−0.26	0.25	0.61†	0.13	0.55*
TF	0.94†		−0.62†	0.37	−0.29	0.21	0.55*	0.34	0.54*
Activity	−0.60†	−0.58†		−0.49*	0.46*	−0.40	−0.59†	−0.07	−0.79†
TC/HDL	0.25	0.24	−0.02		−0.77†	0.04	0.15	0.36	0.49*
Log HDL	−0.18	−0.15	0.13	−0.49*		0.16	−0.29	−0.39	−0.62†
FPG	0.31	0.25	−0.24	−0.10	0.16		0.17	−0.49*	0.16
Log G_{120}	0.31	0.40	−0.10	0.11	−0.48*	−0.10		0.06	0.72†
Log FPI	0.29	0.39	0.11	0.44	−0.18	−0.06	0.25		0.29
Log I_{120}	0.26	0.29	−0.19	0.16	−0.42	−0.17	0.37	0.37	

NOTE. Correlations for SCI are in the top right of the matrix and controls in the bottom left of the matrix.

Abbreviations: Fat %, percentage of body fat; TF, trunk fat mass; TC/HDL, ratio of total cholesterol to high-density lipoprotein cholesterol (HDL); FPG, fasting plasma glucose; G_{120}, 2-hour glucose value; FPI, fasting plasma insulin; I_{120}, 2-hour insulin value.

*$P < .05$.

†$P < .01$.

ations were observed between activity and adiposity measures; and in HDL with TC/HDL and postload glucose.

Using our criteria for the presence of the metabolic syndrome, 11 of the 20 SCI group had IGT and/or hyperinsulinemia. Of this group, 5 were both obese and dyslipidemic, while the remaining 6 met 1 of these 2 additional criteria. IGT was not observed in any of the controls.

Factor Analysis

A large number of correlations lay above the recommended cut-off of 0.3.[6] The Kaiser-Meyer-Olkin (KMO) measure of sampling adequacy was 0.7 and 0.6 for the SCI and control groups, respectively, and Bartlett's test of sphericity was significant at the $P = .0005$ level for both groups. Furthermore, the average communality score for the SCI group was 0.8 and 0.7 for the control group. A 3-factor model was found for both the SCI and control groups. This model accounted for 80.3% and 69% of the variance in the original 9 variables examined in the SCI and control groups, respectively. The factor loadings for each group are shown in Tables 3 and 4.

In the SCI group, the first factor accounted for 37% of the variance in the total data set and was characterized by positive factor loadings of ≥0.4 on fat percent, trunk fat, postload insulin, and glucose. Activity levels negatively loaded on this factor. Factor 2 accounted for 26.2% of the variance in the total data set and was characterized by positive loadings on TC/HDL and postload insulin. Negative factor loading was observed for both HDL and activity. The third factor accounted for 17.1% of the variance in the total data set and was characterized by a positive loading on fasting plasma insulin and a negative loading on fasting glucose. Thus, factor 1 reflects the interaction between total adipose tissue, fat distribution, and postload carbohydrate (CHO) handling, with lower activity levels associated negatively with these variables. Factor 2 reflects dyslipidemia and insulin interaction, with lower activity again interacting negatively with these variables, while factor 3 is interpreted as a postabsorptive state.

Factor 1 in the control group accounted for 28.6% of the variance in the total data set and was characterized by positive loading on fat percent, trunk fat, and fasting plasma glucose. Activity negatively loaded on factor 1. The second factor accounted for 22.2% of the variance in the total data set and was characterized by positive loading on postload insulin and glucose. Negative loadings for factor 2 were observed on HDL and fasting plasma glucose. The third factor in the control group accounted for 18.2% of the variance in the total data set

Table 3. Factor Loadings After Orthogonal Rotation of Original Variables in the SCI Group

Variable	Factor 1	Factor 2	Factor 3
Fat percent	**0.92**	0.11	−0.03
Trunk fat mass	**0.90**	0.14	0.11
Activity	**−0.69**	**−0.51**	0.30
TC/HDL	0.15	**0.90**	0.08
Log HDL	−0.16	**−0.90**	−0.22
Fasting glucose	0.25	0.03	**−0.87**
Log 2-hour glucose	**0.79**	0.14	−0.07
Log fasting insulin	0.20	0.30	**0.82**
Log 2-hour insulin	**0.65**	**0.60**	−0.03
Percentage of variance	37.0	26.2	17.1
Cumulative variance (%)	37.0	63.2	80.3

NOTE. Factor loadings >0.4 are in bold type.

Abbreviation: TC/HDL, ratio of total cholesterol to high-density lipoprotein cholesterol (HDL).

Table 4. Factor Loadings After Orthogonal Rotation of Original Variables in the Control Group

Variable	Factor 1	Factor 2	Factor 3
Fat percent	**0.90**	0.17	0.25
Trunk fat mass	**0.88**	0.22	0.29
Activity	**−0.78**	−0.18	0.25
TC/HDL	0.03	0.20	**0.79**
Log HDL	−0.007	**−0.76**	−0.25
Fasting glucose	**0.56**	**−0.46**	−0.02
Log 2-hour glucose	0.21	**0.74**	0.06
Log fasting insulin	0.10	0.16	**0.84**
Log 2-hour insulin	0.15	**0.70**	0.17
Percentage of variance	28.6	22.2	18.2
Cumulative variance (%)	28.6	50.8	69.0

NOTE. Factor loadings >0.4 are in bold type.

Abbreviation: TC/HDL, ratio of total cholesterol to high-density lipoprotein cholesterol (HDL).

and was characterized by positive factor loadings on both fasting plasma insulin and TC/HDL. For the control group, factor 1 is reflective of the interaction between total adiposity and fat distribution and postabsorptive glucose, with a negative interaction of activity. Factor 2 is representative of the interaction between lipid and carbohydrate metabolism, with higher postload glucose and insulin and lower HDL interacting with lower fasting plasma glucose. The interaction between insulin and the atherogenic marker of TC/HDL is reflected in factor 3.

DISCUSSION

The purpose of this study was to investigate the presence of metabolic syndrome, using modifications of the WHO definition and including total physical activity levels (minutes per week), in a group of active males with spinal cord injury who were carefully matched for age, height, and weight with active able-bodied males. While the WHO criteria used to identify the presence of the metabolic syndrome are reliable for the able-bodied population, some recommended values are unsuitable for the SCI population. BMI and blood pressure measurements used in able-bodied populations to identify obesity and hypertension, respectively, have little validity when applied to the SCI population. Using modified criteria to identify the metabolic syndrome in this study, IGT and/or hyperinsulinemia, plus body fat $\geq$ 25% and/or HDL $<$ 1.04 mmol/L, the metabolic syndrome was present in 55% of the SCI group.

Factor analysis revealed 3-factor models for both groups in this study. Three-factor models have been reported by others in able-bodied groups, with various combinations of adiposity measures, lipid or glucose, and insulin values clustering together to determine each factor.[10,11,21,22] It has been proposed that if all the identified variables contribute equally to the etiology of the metabolic syndrome then, in factor analysis, all variables would cluster together under a single factor.[8,11,22,23] However, the use of factor analysis has routinely identified 2-, 3-, and 4-factor models, indicating that a limited number of underlying physiologic abnormalities constitute the characteristics of the metabolic syndrome. Common findings among factor analysis studies are the appearance of 2- to 4-factor models, loading of insulin on more than 1 factor, and a separate factor clustering various blood pressure measures.[8-11,21-24]

In this study, the first factor identified for the SCI group was characterized by positive loadings on a cluster of adiposity measures and indicators of IGT and β-cell function (high postload glucose and insulin values), with activity showing a negative association. Activity was negatively loaded and postload insulin positively loaded in the second factor, along with the indicators for dyslipidemia. The presence of insulin and activity in these 2 factors may be indicative of key contributions of both insulin (positive) and activity (negative) to the development of the metabolic syndrome. Previous factor analysis research has identified insulin resistance and hyperinsulinemia as a unifying theme with defects in insulin action and enhanced second phase insulin release from the β cells of the pancreas contributing to hyperglycemia and hyperinsulinemia.[25] In addition, centrally located adiposity[26] contributes to insulin resistance and hypersecretion of insulin, which has been shown to occur in obese subjects,[27,28] thus leading to the development of the metabolic syndrome.

While physical inactivity has not been recognized as a characteristic of the metabolic syndrome, it does contribute to the development of both diabetes[29] and CHD,[30] in which the risk factors for metabolic syndrome are common. Physical activity enhances insulin sensitivity[31] and contributes to the clearance of glucose from the circulation by a contraction-stimulated, insulin-independent mechanism.[32] Moreover, increased physical activity can reduce FM, including centrally located fat.[33,34] Given the close relationship of physical inactivity with increased adiposity, insulin and glucose responsiveness, the negative loading of activity in this factor is not unexpected. Negative loading of physical activity, in conjunction with body mass measures, has also been reported by Lakka et al.[19]

The second factor in the SCI group was reflective of dyslipidemia. The high atherogenic ratio of TC/HDL and low HDL were associated with high postload insulin and low activity levels. Low levels of HDL are recognized as an independent risk factor for CHD development,[35] while the atherogenic ratio of TC to HDL has been shown to be a better predictor of CHD risk than either of these 2 alone.[36] This atherogenic ratio has also been identified in individuals with a cluster of other metabolic abnormalities, including hyperinsulinemia.[37] Physical activity augments HDL levels, with regular physical activity decreasing TC/HDL levels.[38,39] Elevated insulin levels are known to contribute to the risk of CHD.[40] Furthermore, a link between low HDL levels and hyperinsulinemia has been reported, with insulin resistance being identified as a common defect that provides a link between HDL and insulin.[41] Insulin has been shown to load on more than 1 factor and is also clustered with measures of obesity, insulin sensitivity, insulin resistance, hyperinsulinemia, and IGT.[10,21,22]

The third factor identified by factor analysis in the SCI group was a combination of increased fasting levels of insulin and decreased fasting levels of glucose. This factor is taken to reflect disturbed carbohydrate metabolism in the fasted state in this group. The fact that these 2 variables clustered together independently as a separate factor suggests that regulation of blood glucose in the fasted state is the result of a specific physiologic mechanism. This is in agreement with previous reports that fasting glucose and insulin and postload glucose and insulin load on separate factors, and it has been proposed that this may be reflective of distinct physiologic processes.[42]

In the control group, the first factor was a cluster of increased adiposity and decreased activity, with a positive loading for fasting glucose. Previous reports indicate fasting glucose has been clustered with adiposity measures.[11,21,24,42] For the present control group, fasting glucose was the only variable to load in 2 factors. The second factor in this group was characterized by increased postload glucose and insulin, with decreased HDL and fasting glucose, again this clustering pattern has been previously reported.[11,21,24,42] The third factor was characterized by a cluster of 2 variables, increased fasting insulin and the atherogenic ratio of TC/HDL.

While slightly different variables grouped under each of the 3 factors for the SCI and control groups, a number of similar clusters were identified; all are in agreement with existing literature.[8-11,21,22,24,42,43] Results of factor analyses may vary as the variables selected for inclusion differ, however some consistency has been identified. Activity was included in the

present study because of the close association between levels of activity and known risk factors of diabetes and CHD, including hyperinsulinemia, obesity, and IGT. Physical activity loaded in a similar manner to that reported by Lakka et al.[19]

While the factor analysis procedure is suited to larger sample sizes, there are a number of checks that can be undertaken to ensure the validity and reliability of this procedure. Bartlett's test was significant in both the SCI and control groups, and the KMO measure was above 0.6, considered to be the minimum value for good factor analysis.[6] It is also considered that good recovery in factor analysis is more dependent on the level of communality rather than the sample size or the sample size to number of variables analyzed.[44] Average communalities for both groups were above the desirable level of ≥ 0.7.[44] Furthermore, a conservative loading for interpretation of ≥ 0.4 was set as the acceptable level for inclusion of a given variable in the factor model. Moreover, the present results are in close agreement with those expressed by others, and there is an acceptable level of agreement of variables clustered within each factor between the SCI and control groups. The identification of a 3-factor model for both groups supports the hypothesis that the development of the metabolic syndrome has multiple etiologic abnormalities.[8] The strong association of physical activity with 2 of the 3 factors in the SCI group supports the relationships found between measures of glycemia and insulinemia and the various measures of adiposity.

In summary, factor analysis identified 3-factor models for both the SCI group and controls. The 3 factors for the SCI group were associations between body fat and glucose tolerance, dyslipidemia and insulin resistance, and fasting CHO variables. Results were similar for the 3 factors in the control group. While cluster variables differed slightly for each group, all identified cluster associations are similar to those reported by others. Thus, we suggest that the WHO definition of the metabolic syndrome, with modification of anthropometric and lipid measures, is suitable to identify metabolic syndrome in SCI individuals. Further prospective studies are required to investigate the long-term health significance of the metabolic syndrome once this cluster of risk factors has been identified.

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