## ORAL DISPOSITION INDEX AS A PREDICTOR OF CHANGES IN GLUCOSE TOLERANCE STATUS OVER TIME

<u>Bárbara Limberger Nedel, Leticia Maria Tedesco Silva, Tassia Cividanes Pazinato, Rodrigo Soares de Souza Marques, Leonardo de</u>

Andrade Mesquita, Luciana Pavan Antoniolli, Vanessa Piccoli, Mayara Beer and Fernando Gerchman

Hospital de Clínicas de Po	orto Alegre, Federal	<b>University of Rio Grande</b>	do Sul, Porto Alegre, Brazil

OBJECTIVES	METHODS
Type 2 diabetes (DM) is characterized by both	Longitudinal study
insulin resistance and $\beta$ -cell dysfunction. The oral	• At first evaluation, 179 patients from an outpatient clinic were submitted to a 75g- OGTT and
disposition index (DIo), a measurement of ß-cell	classified according to different degrees of glucose tolerance based on ADA criteria.
function and insulin sensitivity, is considered the best	• Patients with prediabetes (PDM) and DM had their data recollected, and patients were
predictor of progression to a worse glucose tolerance	classified as regressors and non-regressors to a better GTS.
status (GTS), although it was not well tested for	<ul> <li>Insulin sensitivity was estimated as 1/fasting insulin and β-cell function as the ratio of the</li> </ul>

regression to a better GTS.

Therefore, the objective is to assess the validity of DIo in our population and to determine whether DIo predicts regression to a better GTS.

change in insulin to the change in glucose from 0 to 30 ( $\Delta$ I0–30/ $\Delta$ G0–30). The DIo was calculated as  $(\Delta IO - 30/\Delta GO - 30 \times 1/fasting insulin)$ .

• Data are presented as mean ± standard deviation (SD), median (P25-P75), unless otherwise specified. A two-sided P value < 0.05 was considered significant.

	SUBJECTS'S CHAR EVA		BASAL AND FINAL CHARACTERISTICS OF THE RE-EVALUATED SUBJECTS									
	NGT (n = 32)	PDM (n = 76)	DM (n = 71)		Ρ			R	egressors (n = 45)	Non-regressors (n = 56)	Ρ	
Age (years)	46.7 ± 12.9	54.8 ± 11.5	53.2 ± 11.2	0	.005 Age	(years)		Ę	57.1 ± 11.6	53.1 ± 11.2	0.084	
Female - n (%)	23 (71.9)	57 (75)	44 (62)	0	.850 Fem	ale - n (%)			32 (71.1)	39 (69.9)	0.874	
DM family history - n (%)	12 (41.4)	32 (43.2)	33 (61.1)	0	.220 BMI	(kg/m²)			33 ± 6.1	$36.5 \pm 6.8$	0.725	
Years of eduction	10 (6.3 – 13.7)	7 (4.2 – 11)	7(4.2 - 11) $7(4 - 11)$ $0.2$		0.263 Wais	Waist circumference (cm)			05.9 ± 13.9	108.3 ± 12.7	0.369	
Sedentarism - n (%)	13 (50)	31 (48.4)			.138 Fast	Fasting glucose (mg/dL)			(100 – 118.5)	108 (97.5 – 115.5)	0.12	
BMI (kg/m <sup>2</sup> )	29.8 (25.5 – 32.5)	30.5 (27.4 – 35.8)	(27.4 – 35.8) 32 (27.6 – 36.2) 0.072		.072 2-ho	our glucose (m	ng/dL)	202 (	161.5 - 240.5	5) $170(157 - 219.5)$	0.184	
Waist circumference (cm)	96 ± 14.8	103.6 ± 12.7	$3.6 \pm 12.7$ 105.4 ± 13.2 0.005		.005 A1c	(%)		6.	1 (5.7 – 6.6)	6.1 (5.7 – 6.9)	0.862	
A1c (%)	5.3 (5 – 5.5)	5.9 (5.5 – 6.2)	(5.5 - 6.2) 6.7 (6 - 7.4)		- Time	Time of follow-up (months)			6 (16 – 43.5)	29 (19 – 37)	0.75 <sup>°</sup>	
Fasting glucose (mg/dL)	87 (82 – 92)	101 (90 – 107)	l (90 – 107) 110 (99 – 124.5)		- Fina	IA1c (%)		5.	6 (5.4 – 5.9)	6 (5.4 – 6.6)	<0.00	
2-hour glucose (mg/dL)	110 (96 – 121)	152.5 (118.7 – 171)	211 (172.5 – 2	66)	- Fina			95	(90 - 108.5)	115 (103 – 128)	<0.00	
$\Delta I / \Delta G$ (uIU/mL per mg/dI)	1.4 (0.8 – 1.8)	1 (0.6 – 1.6)	0.5 (0.3 – 1)	)		Final BMI (kg/m <sup>2</sup> )			31.4 ± 5.8	33.1 ± 7.5	0.26	
HOMA-S (%)	105.4 (63.8 – 196.6)	80.6 (55.1 – 120.5)	68.4 (45.5 – 91	1.9)	- Fina	Final waist circumference (			3 (90 – 112)	106 (94.5 – 110.5)	0.42	
HOMA-IR (%)	1.9 (1 – 3.1)	2.5 (1.7 – 3.9)	3.5 (2.6 – 5.4	1)	<b>D</b> .		(7.00000)	0.0	(00001)	0.7 (-0.9 – 1)	<0.00	
			``	,	- ULTS	osition index	(Z SCOre)	-U.C	6 (-0.9 – -0.1)	0.7 (-0.3 - 1)		
Is the Oral Disp	osition Index valid in opulation?	ls the β-c	``	RESI	ULTS predict the re	gression				n and progression	rates?	
Is the Oral Disp our p		Is the β-c to	cell function a better gluc	RESU able to provide to ble to provide to pr	ULTS predict the re erance status	egression s?	<b>Which</b> •			n and progression	rates?	
Is the Oral Disp our p		ls the β-c	cell function a a better gluc	RESI	ULTS predict the re	gression	Which • 80 • 70 •		e regressio	n and progression	rates?	
Is the Oral Disp our p	opulation?	Is the β-c to	ell function a better gluc	RESU ble to pose tol Hazard	ULTS predict the re erance status	egression s?	Which         80         70         20 </td <td></td> <td>e regressio</td> <td>n and progression Basal PDM Basal DM (1 43.6%</td> <td><b>rates?</b> (n=46) n=55)</td>		e regressio	n and progression Basal PDM Basal DM (1 43.6%	<b>rates?</b> (n=46) n=55)	
Is the Oral Disp our p	opulation? • NGT	Is the β-c to Indepen variab	ell function a better gluc	AESU ble to pose tol Hazard ratio	ULTS predict the re erance status	egression s? P	Which         80         70         70         90         90         50	were the	e regressio	n and progression Basal PDM Basal DM (1 43.6%	<b>rates</b> (n=46) n=55) 36.4%	
Is the Oral Disp our p	<ul> <li>NGT</li> <li>PDM</li> </ul>	Is the β-c to Indepen variab Sex	cell function a better gluc dent Beta les -0.055 0.036	RESU ble to ose tol Hazard ratio 0.945	ULTS predict the re erance status IC (95%) 0.448 – 2.003	egression s? P 0.886	Which         80         70         70         70         60         90         10 </td <td></td> <td>e regressio 56.5% (n=26)</td> <td>n and progression Basal PDM Basal DM (<math>a</math> 43.6% (n=24) 20% (n=11)</td> <td><b>rates</b> (n=46) n=55)</td>		e regressio 56.5% (n=26)	n and progression Basal PDM Basal DM ( $a$ 43.6% (n=24) 20% (n=11)	<b>rates</b> (n=46) n=55)	
Is the Oral Disp our p	<ul> <li>NGT</li> <li>PDM</li> </ul>	Is the β-c to Indepen variab Sex Age Waist	Cell function a better gluc dent Beta les -0.055 0.036	AESU ble to good and a second a	ULTS predict the re erance status IC (95%) 0.448 – 2.003 1.003 – 1.072	egression s? P 0.886 0.032	Which         80         70         70         70         80         70         40	were the 28.3%	e regressio 56.5% (n=26)	n and progression Basal PDM Basal DM ( $a$ 43.6% (n=24) 20% (n=11)	<b>rates</b> (n=46) n=55) 36.4%	
Lis the Oral Disp our p	<ul> <li>NGT</li> <li>PDM</li> </ul>	Is the β-c to Indepen variab Sex Age Waist circumfer DM family	Cell function a better gluc dent es -0.055 0.036 0.036 0.036 0.036 0.036 0.036	AESU ble to ose tol Hazard ratio 0.945 1.037 0.985	ULTS predict the re erance status IC (95%) 0.448 – 2.003 1.003 – 1.072 0.961 – 1.009	egression ? P 0.886 0.032 0.225	Which         80         70 </td <td>were the 28.3%</td> <td>e regressio 56.5% (n=26)</td> <td>n and progression Basal PDM Basal DM (<math>a</math> 43.6% (n=24) 20% (n=11)</td> <td><b>rates?</b> (n=46) n=55) 36.4%</td>	were the 28.3%	e regressio 56.5% (n=26)	n and progression Basal PDM Basal DM ( $a$ 43.6% (n=24) 20% (n=11)	<b>rates?</b> (n=46) n=55) 36.4%	
Lis the Oral Disp our p	<ul> <li>NGT</li> <li>PDM</li> <li>DM</li> </ul>	Is the β-α to Indepen variab Sex Age Waist circumfer DM family history	Cell function a better gluc dent es -0.055 0.036 0.036 0.036 0.036 0.036 0.036	<b>RESU Ble to good </b>	ULTS predict the re erance status IC (95%) 0.448 – 2.003 1.003 – 1.072 0.961 – 1.009 0.134 – 1.382	egressions? P 0.886 0.032 0.225 0.157	Which         80         70 </td <td>were the 28.3%</td> <td>e regressio 56.5% (n=26) 15.2 (n= 200 200 200 200 200 200 200 200 200 20</td> <td>n and progression Basal PDM Basal DM (n 43.6% (n=24) 20% (n=11) 1 1 1 1 1 1 1 1 1 1 1 1 1</td> <td><b>rates?</b> (n=46) n=55) 36,4%</td>	were the 28.3%	e regressio 56.5% (n=26) 15.2 (n= 200 200 200 200 200 200 200 200 200 20	n and progression Basal PDM Basal DM (n 43.6% (n=24) 20% (n=11) 1 1 1 1 1 1 1 1 1 1 1 1 1	<b>rates?</b> (n=46) n=55) 36,4%	

	NGT (n = 32)	PDM (n = 76)	DM (n = 71)	Ρ	2					R	egresso (n = 45)		U	ressors 56)	Ρ
Age (years)	46.7 ± 12.9	54.8 ± 11.5				Age (years)				57.1 ± 11.6			53.1 :	0.084	
Female - n (%)	23 (71.9)	57 (75)				Female - n (%)					32 (71.1	)	39 (6	0.874	
DM family history - n (%)	12 (41.4)	32 (43.2)			20	<ul> <li>BMI (kg/m<sup>2</sup>)</li> <li>Waist circumference (cm)</li> <li>Fasting glucose (mg/dL)</li> <li>2-hour glucose (mg/dL)</li> <li>A1c (%)</li> <li>Time of follow-up (months)</li> </ul>				33 ± 6.1		$36.5 \pm 6.8$		0.725	
Years of eduction	10 (6.3 – 13.7)	7 (4.2 – 11)			263				105.9 ± 13.9			108.3 ± 12.7		0.369	
Sedentarism - n (%)	13 (50)	31 (48.4)			38				106	(100 – 1	18.5)	108 (97.5 – 115.5)		0.128	
BMI (kg/m²)	29.8 (25.5 – 32.5)	30.5 (27.4 – 35.8)	27.4 – 35.8) 32 (27.6 – 36.2)		)72				202 (	161.5 – 2	240.5)	170 (157 – 219.5)		0.184	
Waist circumference (cm)	96 ± 14.8	103.6 ± 12.7	8.6 ± 12.7 105.4 ± 13.2		05				6.1	1 (5.7 – 6	6.6)	6.1 (5.7 – 6.9)		0.862	
A1c (%)	5.3 (5 – 5.5)	5.9 (5.5 – 6.2)	5.5 – 6.2) 6.7 (6 – 7.4)		-			26 (16 – 43.5)			29 (19 – 37)		0.751		
Fasting glucose (mg/dL)	87 (82 – 92)	101 (90 – 107)	) – 107) 110 (99 – 124.5)		-	Final A1c (%)		5.6 (5.4 – 5.9)			6 (5.4 – 6.6)		<0.001		
2-hour glucose (mg/dL)	110 (96 – 121)	152.5 (118.7 – 171)	211 (172.5 – 266) -		-	Final fasting glucose (mg/dL)			.)	95 (90 – 108.5)			115 (103 – 128)		<0.001
$\Delta I / \Delta G$ (uIU/mL per mg/dI)	1.4 (0.8 – 1.8)	1 (0.6 – 1.6)	1.6) 0.5 (0.3 – 1)		-	Final BMI (kg/m <sup>2</sup> )				31.4 ± 5.8			33.1 ± 7.5		0.265
HOMA-S (%)	105.4 (63.8 – 196.6)	80.6 (55.1 – 120.5)	1 – 120.5) 68.4 (45.5 – 91.9)		-	Final waist circumference (cm)			cm)	103 (90 – 112)			106 (94.5	5 – 110.5)	0.429
HOMA-IR (%)	1.9 (1 – 3.1)	2.5 (1.7 – 3.9)	3.5 (2.6 – 5.4	+) -	-	Disposition ir				-0.8 (-0.90.1)		-0.1)	0.7 (-0	.9 – 1)	<0.001
-	sition Index valid in pulation?		ell function a a better gluc					Whi	ch w	ere the	e regres	ssion a	and prog		
2,5 (10) 2	• NGT	Indepen variab	Вета	Hazard ratio	IC (95°	%)	Ρ		80 70 - 60 -		56.5% (n=26)			Basal PDM ( Basal DM (n <sup>:</sup>	•
	PDM	Sex	-0.055	0.945 0	).448 – 2	2.003	0.886	(%) e			(			43.6%	
<b>b</b> 1,5	◆ DM	Age	0.036	1.037 1	.003 – 1	1.072	0.032	rate	50 -	20 20/					36.4% n=20)
nlU/mL 1		Waist circumfer	-0.015 ence	0.985 0	).961 – 1	1.009	0.225	atients	40 - 30 - 20 -	28.3% (n=13)		15.2%	20% (n=11)		
<b>5</b> <b>5</b> <b>7</b> <b>9</b> <b>7</b> <b>9</b> <b>9</b> <b>9</b>		DM family history	-0.842	0.431 0	).134 – 1	1.382	0.157	Å.	20 - 10 -			(n=7)			
		SD DIo *	-0.428	0.652 0	).451 – 0	).943	0.023		0 +						
0 0,05 0,1	0,15 0,2 0,25 0,3	0,35								NGT	PDM	DM Fir	NGT nal GTS	PDM	DM
1/ins [(ulU/mL)⁻¹]		* SD DIo:	* SD DIo: DIo adjusted by Z score									E II			

## CONCLUSIONS

## References

A total of 47.5% of patients regressed to a better glucose tolerance status while participating in a program with multiple interventions for the treatment of hyperglycemia. The Oral Disposition Index in our population was able to predict the regression to a better glucose tolerance status. It was proved to be reproducible and could be applied for DM research in the Brazilian population. Our outpatient clinic presents regression and progression rates of the glucose tolerance statuts compatible with epidemiologic studies.

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