Diabetes & Metabolic Syndrome: Clinical Research & Reviews 13 (2019) 716–720

Contents lists available at ScienceDirect

Diabetes & Metabolic Syndrome: Clinical Research & Reviews

journal homepage: www.elsevier.com/locate/dsx

Perspective

High rates of atherogenic dyslipidemia, β-cell function loss, and microangiopathy among Turkish migrants with T2DM

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Keywords:
Type 2 diabetes
Turk
Atherogenic dyslipidemia
β-cell function
Microangiopathy

A B S T R A C T

Aims: Non-Caucasian migrants require dedicated approaches in diabetes management due to specific genetic; socio-cultural; demographic and anthropological determinants. Documenting such phenotypes allows for better understanding unmet needs and management priorities.
Methods: This age- and sex-adjusted case-control (1:6 ratio) study compared 56 T2DM Turkish migrants (70% males) resident in Belgium [Tu] with 336 T2DM Caucasians [Ca], all benefiting from state-funded healthcare.
Results: The 2 groups did not differ regarding BMI; waist circumference; fat mass; visceral fat; muscle mass; insulin sensitivity; insulinemia; metabolic syndrome; hypertension; lipid-modifying drugs; and macroangiopathy. They also had similar renal function and (micro)albuminuria. Education (low/high) and ethanol consumption were lower among [Tu]: 83/17% and 2.0 U/wk vs 43/57% and 13.6 U/wk in [Ca] ($p < 0.0001$). β-cell function loss (BCF) was higher in [Tu]: 1.58(0.45) vs 1.35(0.54)%/yr (p 0.0027), as was HbA1c: 8.39(1.91) vs 7.48(1.35)% in [Ca] ($p < 0.0001$). Diabetes duration and insulin use were increased in [Tu]: 19(9)yr and 70% vs 16(8)yr and 48% in [Ca] (p 0.0111 and 0.0024). Atherogenic dyslipidemia (AD) was more prevalent in [Tu]: 64% vs 49% (p 0.0309), who had higher non-HDL-C; apolipoprotein B_{100}; LDL-C; and triglycerides; and lower HDL-C and apolipoprotein A-I levels (all $p < 0.05$). Overall microangiopathy; retinopathy; and neuropathy were more prevalent in [Tu]: 55-35-37% vs 40-18-20% in [Ca] (all $p < 0.05$).
Conclusions: These results should raise concerns about poor glycaemic control; rapid BCF loss; severe AD; and microangiopathy among Turkish migrants with T2DM. Targeting AD could improve the cardiometabolic profile of this minority given the relationship between AD and residual vascular risk.

1. Introduction

Ethnic minorities living in Europe show an increased prevalence of type 2 diabetes mellitus (T2DM) compared to host inhabitants. Management of T2DM in ethnic minorities requires specific approaches, although the general recommendations for the treatment of chronic hyperglycemia are identical regardless of ethnogeographic origin. In addition to obvious socio-cultural differences among persons from minorities, that can have positive or negative impacts on the care required for chronic diseases, there are intrinsic characteristics that modify their cardiometabolic phenotype when some of them develop T2DM [1,2].

In Belgium, ethnic minorities are mainly composed of 1st or 2nd generation economic migrants from Morocco, sub-Saharan Africa, and Turkey. We previously documented the many specificities of T2DM patients from the Maghreb and sub-Saharan Africa, in whom we observed phenotypic differences in numerous cardiometabolic variables. As a follow-up to these studies, we wanted to document Turkish patients with T2DM resident in Belgium, to see if there are also substantial differences [3–5]. Turkish populations, historically native to Central Asia, settled in the Anatolian basin and have to this day preserved a partially Asian genotype-phenotype, an

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https://doi.org/10.1016/j.dsx.2018.11.049
1871-4021/

inheritance that also concerns migrants [6]. Economic migrants being relatively young, low-skilled men with non-sedentary occupations, they should *a priori* represent a low-risk population for incident T2DM. Yet, this is not what is observed in real-life for prevalent/incident T2DM, suggesting that some adverse determinants in terms of carbohydrate homeostasis are at play among them.

The purpose of this case-control study was to document in depth the cardiometabolic phenotype of Turkish T2DM patients from Belgium (vs European Caucasians controls) in order to identify any characteristics that could modulate their diabetes phenotype and/or be relevant for individualization of care in such population.

2. Patients & methods

This cross-sectional case-control study analysed the medical records of T2DM patients from 1st generation Turkish migrants residing in Belgium (cases; n = 56; [Tu]). They were compared to age- and gender-matched European Caucasians with T2DM living in Belgium (n = 336; [Ca]), in a «1 case-to-6 controls» ratio. European Caucasians were distributed as follows: Belgians (81.6%); Italians (6.8%); Germans (2.1%); Spanish-Portuguese (2.1%); French (1.5%); British (1.2%); and other Western European nationals (5.7%). All Turkish and Caucasians were followed as diabetic outpatients at a single academic hospital in Brussels, and benefited from the Belgian National healthcare or from the European Union social security to cover the costs of diabetes care, including purchase of medicines and self-monitoring of blood glucose.

Exclusion criteria included patients treated with medications that could affect insulin sensitivity (IS) or β-cell function (BCF), other than glucose-lowering drugs, including systemic or topical corticosteroids, antiretrovirals, immune-modulators, and antipsychotics. Were also excluded patients with chronic inflammatory or infectious diseases, ongoing infections and/or other acute conditions, cancer or major organ failure (respiratory, heart, and liver). Each patient gave informed consent, and the study was performed in agreement with the principles of the Declaration of Helsinki and Good Clinical Practice, and the Institutional Review Board.

The following sociodemographic and clinical variables were recorded: age; gender; age at diabetes diagnosis; diabetes duration; education (as proxy for socio-economic status); smoking; ethanol intake; recreational physical activity; weight; height; waist circumference; body mass index (BMI); blood pressure (BP); fat mass, visceral fat and skeletal muscle mass (Omron BF 500, Omron Healthcare Europe B.V., Hoofddorp, The Netherlands). First-degree (mother and/or father and/or siblings) familial histories for DM and/or early-onset coronary heart disease (EOCHD), defined by the occurrence of a 1st CHD event <55 years (men) and <65 years (women), were also recorded. Hypertension was defined as systolic BP ≥ 140 and/or diastolic BP ≥ 90 mmHg, as well as ongoing antihypertensive medication(s). Non-alcoholic hepatic steatosis was considered in case of ultrasonic hyper-reflectivity and in the absence of etiological factors associated with fatty liver, including excess ethanol intake.

Ongoing therapies were recorded, including glucose-lowering drugs (metformin; sulfonylureas/glinides; thiazolidinediones; dipeptidyl peptidase-4 inhibitors; sodium-glucose transporter-2 inhibitors; glucagon-like peptide-1 receptor agonists; insulin); BP-lowering therapies; lipid-modifying drugs (LMDs: statins; fibrates and/or ezetimibe); and aspirin.

Methods used to define the presence of a metabolic syndrome (MetS), atherogenic dyslipidemia (AD), microangiopathies (diabetic retinopathy; peripheral neuropathy), and macrovascular complications [coronary artery disease; transient ischemic attack (TIA)/stroke; peripheral artery disease] were previously described [7–9]. BCF and IS were assessed with HOMA-2 modelling. Values of insulin secretion ([B]; normal 100%) were plotted as a function of IS ([S]; normal 100%), defining a *hyperbolic product* area [BxS] (unit: $\%^2$; normal: 100%, corresponding to $10^4\%^2$), representing residual β-cell function. Secular loss of hyperbolic product ([BxS] loss rate; $\%.year^{-1}$) was obtained by dividing (100%-[BxS]) by patient's age [3,4,10,11].

HbA1c, fasting glucose and insulinemia were determined by routine methods. The hyperglycemia index was calculated as the product between the amount of HbA1c exceeding the upper normal value of 42 mmol/mol and duration of diabetes. Fasting lipids and lipoproteins (total cholesterol (C), HDL-C, triglycerides (TG); LDL-C (calculated using Friedewald's formula), non-HDL-C, lipoprotein(a), and apolipoprotein B_{100}) were analysed by routine methods. Microalbuminuria was considered for urine albumin (mg/dL)/creatinine (g/dL) > 30 mg/g.

Results are presented as means (±1 standard deviation; SD) or proportions (%). The significance of differences between means was assessed using Student's *t*-test, or Welch's test for data sets with significant differences in SDs, and using Fisher's Exact test for differences in proportions. Results were considered statistically significant or non-significant (NS) for $p < 0.05$ or $p \geq 0.05$, respectively.

3. Results

Mean age of [Tu] was 66 (12) years, and gender ratio (men-women) was 70%–30% (Table 1). A family history of diabetes was significantly more often reported by [Tu] patients (+39% relative frequency), while EOCHD familial histories did not differ between groups. The educational level was significantly lower among [Tu], of whom 17% obtained a higher education diploma, compared to 57% among [Ca]. This gap in educational level was more pronounced among women. The age at diagnosis of T2DM was much earlier, by an average of 5 years, among [Tu], whose mean duration of diabetes was increased by an average of 3 years compared to controls.

With regard to lifestyle, smoking was significantly more prevalent among [Tu], while alcohol consumption was significantly higher among [Ca]. The absence of any form of recreational physical activity was frequent in both groups, but significantly more common among [Tu] (+25%). As regards anthropometry, [Tu] patients weighed on average 3.8 kg less than Caucasians, but due to lower height (−5 cm on average), the BMIs of the 2 groups were not different, nor were waist circumference, skeletal muscle mass, fat mass, and visceral fat (Table 1). A MetS was present at high frequency in both groups, with no significant difference between [Tu] and [Ca]. Hypertension prevalence, systolic BP and eGFR were also similar between cases and controls. BP-lowering drugs and aspirin use were not different between groups (*not shown*). Although there was no difference in terms of hepatic steatosis for either sex combined, there was a significantly increased frequency of this comorbidity in Turkish women, all of whom had it (compared to 70% in [Ca]).

IS was reduced to the same extent in both groups, which had similar fasting insulin levels (Table 2). Residual insulin secretion, represented by the hyperbolic product [BxS], was low in both groups, being reduced by almost three-quarters. It was even more reduced, by a relative −16%, among Turks, albeit the difference did not reach significance. Annual BCF loss rate was considerably and significantly increased in [Tu], by 17% for either sex combined, and even more so in Turkish men, in whom the BCF loss rate was increased by a relative 20% in comparison to either [Tu] women or [Ca] men.

Mean HbA1c was higher, by a relative 17%, among [Tu]. The glycemic control of most patients was above the recommended

Table 1
Patients' characteristics.

		Turks	Caucasians	*P*
n		***56***	***336***	~
age	years	**66** (12)	**66** (9)	~
males: females	%	**70: 30**	**70: 30**	~
family history of diabetes	%	**71**	**51**	*0.0056*
family history of EOCHD	%	**8**	**12**	*NS*
education - all	%	**83: 17**	**43: 57**	*<0.0001*
education - males	%	**78: 22**	**38: 62**	*<0.0001*
education - females	%	**94: 6**	**54: 46**	*0.0012*
age at diabetes diagnosis	years	**45** (10)	**50** (10)	*0.0006*
diabetes duration	years	**19** (9)	**16** (8)	*0.0111*
current smokers	%	**27**	**15**	*0.0307*
ethanol	$U.week^{-1}$	**2.0** (5.0)	**13.6** (20.2)	*<0.0001*
no physical activity	%	**79**	**63**	*0.0232*
height	m	**1.68** (0.10)	**1.73** (0.09)	*0.0002*
weight	kg	**85.5** (16.9)	**89.3** (20.1)	*NS*
body mass index	$kg.m^{-2}$	**30.4** (6.0)	**29.9** (6.1)	*NS*
waist circumference	cm	**104** (13)	**106** (16)	*NS*
skeletal muscle mass	%	**32** (4.2)	**31** (4.6)	*NS*
fat mass	%	**30** (8.2)	**31** (9.3)	*NS*
visceral fat	0–30	**12** (4)	**13** (5)	*NS*
metabolic syndrome	%	**91**	**84**	*NS*
hepatic steatosis - all	%	**76**	**78**	*NS*
hepatic steatosis - males	%	**69**	**81**	*NS*
hepatic steatosis - females	%	**100**	**70**	*0.0061*
systolic BP	mm Hg	**141** (21)	**138** (18)	*NS*
hypertension	%	**91**	**93**	*NS*
eGFR	$mL.min^{-1}.1.73\ m^2$	**83** (32)	**76** (25)	*NS*

Results are expressed as means (1 SD) or proportions (%). BP: blood pressure; eGFR: estimated glomerular filtration rate; EOCHD: early-onset coronary heart disease; NS: not significant.

Table 2
Glucose homeostasis.

		Turks	Caucasians	*P*
n		***56***	***336***	~
insulinaemia	$pmol.L^{-1}$	**115** (92)	**114** (84)	*NS*
insulin sensitivity	%	**54** (35)	**55** (42)	*NS*
HOMA Product [B x S]	%	**23.3** (15.6)	**27.8** (19.3)	*NS*
[B x S] loss rate - all	$\%.yr^{-1}$	**1.58** (0.45)	**1.35** (0.54)	*0.0027*
[B x S] loss rate - males	$\%.yr^{-1}$	**1.65** (0.48)	**1.38** (0.53)	*0.0031*
[B x S] loss rate - females	$\%.yr^{-1}$	**1.38** (0.27)	**1.28** (0.54)	*NS*
HbA_{1c}	$mmol.mol^{-1}$	**68** (9)	**58** (7)	*<0.0001*
$HbA_{1c} < 53$ mmol mol^{-1}	%	**22**	**40**	*0.0106*
hyperglycemia index	$mmol.mol^{-1}.year^{-1}$	**430** (475)	**215** (249)	*0.0016*
BCS - metf - TZD - IBT	%	**43–64–2–11**	**41–73–4–22**	*NS*
insulin	%	**70**	**48**	*0.0024*
insulin	$IU.day^{-1}.kg^{-1}$	**0.74** (0.63)	**0.82** (0.67)	*NS*

Results are expressed as means (1 SD) or proportions (%). BCS: beta-cell stimulant; B x S: hyperbolic product between insulin sensitivity and beta-cell function; HbA1c: glycated haemoglobin A1c; IBT: incretin-based therapies; metf: metformin; TZD: thiazolidinedione; NS: not significant.

53 mmol/mol HbA1c in both groups, but failure to achieve therapeutic target was much more often observed among [Tu], at 78% vs. 60% in [Ca]. Cumulative exposure to hyperglycemia over time, estimated by the hyperglycemia index, was twice as high among [Tu] than [Ca]. Ongoing non-insulin glucose-lowering therapies did not differ between groups, although there were twice as many patients treated with IBT in [Ca]. Insulin was significantly more used by Tu (+22% absolute and +46% relative increases), while daily insulin doses were similar to those of [Ca].

There were no statistical differences between groups regarding use of statins, fibrates or ezetimibe (Table 3). Although the daily atorvastatin-equivalent dose was similar between [Tu] and [Ca], LDL-C was higher in [Tu] (+15%), as were non-HDL-C (+16%) and $apoB_{100}$ levels (+11%; all $p < 0.05$). [Tu] patients were also characterized by a very high prevalence (+31%) and a more pronounced severity of AD, which resulted in TG increased by 30%; and HDL-C decreased by 14%. Lipoprotein(a), on the other hand, did not differ between groups.

Regarding all-cause macroangiopathy; CHD; cerebrovascular disease; and peripheral arterial disease, there were no difference in prevalence between [Tu] and [Ca] (Table 4). By contrast, there was a marked and significant increase in the frequency of all-cause microangiopathy (+37%); non-proliferative and proliferative retinopathy (+101%); non-proliferative retinopathy (+100%); and peripheral polyneuropathy (+83%). (Micro)albuminuria was non-significantly more frequent in [Tu].

4. Discussion

This study aimed at analyzing the diabetic phenotype of migrants from Turkey residing in Belgium. Due to earlier onset of hyperglycaemia, any characterization of diabetes in such migrants should take into account, as epidemiologic constrain, the need to compare their phenotype with that of age- and gender-normalized controls. Our data highlight hitherto unknown cardiometabolic characteristics that are all the more relevant that CV risk profile

Table 3
Lipids and lipoproteins.

		Turks	Caucasians	*P*
n		***56***	***336***	~
anti-dyslipidemic drug(s)	%	**77**	**86**	*NS*
statin	%	**70**	**80**	*NS*
ezetimibe	%	**11**	**17**	*NS*
fibrate	%	**14**	**23**	*NS*
atorvastatin-equivalent dose	mg/day	**23** (20)	**20** (17)	*NS*
cholesterol	$mg.dL^{-1}$	**166** (43)	**157** (37)	*NS*
non-HDL-C	$mg.dL^{-1}$	**124** (45)	**107** (36)	*0.0091*
apolipoprotein B_{100}	$mg.dL^{-1}$	**93** (30)	**84** (23)	*0.0358*
LDL-C	$mg.dL^{-1}$	**87** (35)	**76** (30)	*0.0136*
lipoprotein *(a)*	$nmol.L^{-1}$	**50** (97)	**49** (63)	*NS*
HDL-C	$mg.dL^{-1}$	**42** (11)	**49** (17)	*0.0001*
apolipoprotein A-I	$mg.dL^{-1}$	**137** (24)	**146** (31)	*0.0150*
triglycerides	$mg.dL^{-1}$	**215** (214)	**166** (141)	*0.0106*
atherogenic dyslipidemia	%	**64**	**49**	*0.0309*

Results are expressed as means (1 SD) or proportions (%). C: cholesterol; HDL-C: high-density lipoprotein cholesterol; LDL-C: low-density lipoprotein cholesterol; NS: not significant.

Table 4
Micro- and macrovascular complications.

		Turks	Caucasians	*P*
n		***56***	***336***	~
microangiopathy	%	**54.5**	**39.9**	*0.0400*
retinopathy	%	**35.2**	**17.5**	*0.0035*
non-proliferative retinopathy	%	**33.3**	**16.6**	*0.0051*
proliferative retinopathy	%	**1.9**	**0.9**	*NS*
peripheral polyneuropathy	%	**37.0**	**20.2**	*0.0089*
(micro)albuminuria	%	**43.0**	**32.0**	*NS*
macroangiopathy	%	**28.6**	**32.4**	*NS*
coronary artery disease	%	**19.6**	**23.5**	*NS*
cerebrovascular disease	%	**10.7**	**7.7**	*NS*
peripheral artery disease	%	**3.6**	**7.1**	*NS*

Results are expressed as proportions (%). NS: not significant.

comparisons between Turkish and autochthonous Caucasians are scarce [12].

Although ethanol consumption was lower in Turks, the results clearly show that these patients had many unhealthy characteristics, modifiable or not, in socio-demographic, cardiometabolic, lipidic and target-organ damage terms. In particular, a much lower level of education (not unexpected given that subjects were mostly economic migrants), highly prevalent smoking habits, increased sedentarity, earlier onset of diabetes, and poor glycemic control, the latter conducive to increased duration and severity of hyperglycemia. Although there was no difference between Turks and Caucasians in anthropometry and body composition (apart from lower height), there was a sexual dimorphism among Turks in hepatic steatosis, the latter present in all women.

Despite similar insulinemia and IS between Turks and Caucasians, the former showed enhanced loss of BCF, particularly men, such an acceleration likely underlying the earlier onset of diabetes, the more frequent use of insulin, and the poorer metabolic control. Thus, HbA1c was significantly increased among Turks, of whom only one in five met the HbA1c target of 7% (53 mmol/mol). This, combined with earlier onset and longer duration of diabetes, resulted in twice as much exposure to chronic hyperglycemia (vs Caucasians) as shown by the cumulative hyperglycemia index. HOMA modelling data clearly demonstrate that poor glycemic control was related to intrinsic BCF defects rather than to differences in insulin resistance. Expectedly from the above, the prevalence of all-cause microangiopathies and that of organ-specific microvascular damage (retina and peripheral nerves) were significantly increased among Turks.

Whereas there were no differences in lipid-lowering treatment or atorvastatin-equivalent dose between groups, Turkish patients had a worsened lipid profile as regards LDL-C, non-HDL-C, $apoB_{100}$, HDL-C, apoA-I, and triglycerides. Ethnic differences in lipid levels, in particular HDL-C and triglycerides, were reported in the large multiethnic HELIUS study [13]. Our findings imply that Turkish patients have substantial LDL-C hypercholesterolemia together with severe AD. These two comorbidities are frequent in the common form of T2DM, associated with the MetS. Their simultaneous presence considerably increases residual vascular risk, and requires therapeutic intensification since both can be improved by lifestyle changes, increased statin dose, statin permutation, addition of ezetrol, and/or addition of fibrates. The higher prevalence of AD may have contributed to the occurrence of microangiopathic complications, either directly or indirectly, by accelerating BCF decline, thereby aggravating chronic hyperglycemia [9,14,15].

On a practical level, these data allow for considering novel ways of individualizing diabetes care in this minority. The accelerated loss of BCF, especially in men, means that they should be followed more often as outpatients, so that glucose-lowering treatment can be intensified if necessary. Poor glycemic control also needs to be proactively addressed [16]. Smoking cessation counseling should be provided, together with ethnicity-specific nutritional advice [17]. Intensified treatment of hypercholesterolemia and AD should be considered, including medication adherence reinforcement and provision of lifestyle and dietary advice targeting sedentariy and hepatic steatosis, especially in women [18]. Finally, close screening of microvascular complications is required given the higher prevalence of all-cause microangiopathy, retinopathy and neuropathy, comorbid with AD [14,15].

The limits of this study are related to its transversal design, which does not allow to assert causal relationships among the observed differences, nor does it permit to distinguish those related to population genetics from those resulting from epigenetic, environmental or socio-cultural factors. Among the strengths of this study, the case-control design, adjusted for gender and age, was an essential methodological prerequisite. Thus, 1st migration generations are predominantly male and younger than the populations of destination countries, and as a result, the age-pyramid of migrant populations (whether diabetic or non-diabetic) substantially differs from that of host countries. Without such an adjustment, erroneous inferences could be made about the cardiometabolic determinants of ethnic minorities when they subsequently develop T2DM. Although the number of subjects studied was limited, we have very carefully recorded their phenotype, making it possible to identify a series of variables of interest not yet studied. An innovative element from our assessment of carbohydrate homeostasis determinants was the yet unreported finding that an increased annualized loss of BCF predicts earlier onset and more severe hyperglycemia among Turks.

In conclusion, this characterization of diabetes among Turkish migrants in Belgium has shown a high level of unaddressed residual micro- and macrovascular risk linked to accelerated BCF loss, poor glycaemic control; high rate of smoking, residual hypercholesterolaemia and AD. Targeting the latter could improve the cardiometabolic profile of this minority given the relationship between AD, BCF loss, microangiopathies and residual vascular risk.

Acknowledgments

Not applicable.

Abbreviations

AD	atherogenic dyslipidemia
BCF	β-cell function
[BxS]	hyperbolic product between β-cell function and IS
BMI	body mass index
BP	blood pressure
Ca	Caucasian
CHD	coronary heart disease
CV	cardiovascular
CVD	cardiovascular disease
DM	diabetes mellitus
eGFR	estimated glomerular filtration rate
EOCHD	early-onset CHD
HbA_{1c}	glycated hemoglobin
HDL	high-density lipoprotein
HDL-C	high-density lipoprotein cholesterol
HOMA	homeostasis model assessment
IS	insulin sensitivity
LDL	low-density lipoprotein
LDL-C	low-density lipoprotein cholesterol
LMD	lipid-modifying drug
MetS	metabolic syndrome
non-HDL-C	non-high-density lipoprotein cholesterol
NS	non-significant
SD	standard deviation
T2DM	type 2 diabetes mellitus
TG	triglycerides (triacylglycerols)
Tu	Turk

Compliance with ethical standards

Disclosure of potential conflicts of interest

The authors declare that they have no conflict of interest.

Research involving human participants and/or animal

Statement of human rights

For this type of study formal consent is not required.

Statement on the welfare of animals

Not applicable.

Informed consent

Informed consent was obtained from all individual participants included in the study.

The study protocol was approved by the Commission d'Ethique Biomédicale Hospitalo-facultaire de l'Université catholique de Louvain (Bruxelles) B403-2017-16NOV-521.

Availability of data & material

Data are available at the Division of Endocrinology & Nutrition, Cliniques universitaires St-Luc and Institut de Recherche Expérimentale et Clinique (IREC), Université catholique de Louvain, Brussels (Belgium) (*person of contact: Prof MP Hermans*).

Funding

This study received no financial support.

Author's contributions

All authors contributed equally to the manuscript.

All authors read and approved the final version of the manuscript for publication.

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