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1 **VALIDATION OF SEVEN TYPE 2 DIABETES MELLITUS RISK SCORES IN A**
2 **POPULATION-BASED COHORT. THE COLAUS STUDY**

3 Short title: 10-y validation of 7 type 2 diabetes risk scores

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39 interpretation, writing of the report, or decision to submit the article for publication.

40 **DISCLOSURE SUMMARY**

41 The authors report no conflict of interest

42 **ABSTRACT**

43 **Aim:** To assess the validity of seven type 2 diabetes mellitus (T2DM) risk scores in predicting the 10-year
44 incidence of T2DM in a Swiss population-based study.

45 **Methods:** Prospective study including 5131 participants (55% women, age range 35 to 75 years) living in
46 Lausanne, Switzerland. The baseline survey was conducted between 2003 and 2006 and average follow-up was
47 10.9 years. Five clinically-based (Balkau, Kahn clinical, Griffin, Swiss diabetes association and Findrisc) and two
48 clinically and biologically based scores (Kahn CB and Wilson) were tested.

49 **Results:** 405 (7.9%) participants developed T2DM. The overall prevalence of participants at high risk ranged from
50 13.7% for the Griffin score to 43.3% for the Balkau score. Prevalence of participants at high risk among those
51 who developed T2DM ranged from 34.6% for the Griffin score to 82.0% for the Kahn CB score. The Kahn CB score
52 had the highest area under the ROC [value and 95% confidence interval: 0.866 (0.849-0.883)], followed by the
53 Findrisc [0.818 (0.798-0.838)] while the Griffin score had the lowest [0.740 (0.718-0.762)]. Sensitivities and
54 specificities were above 70%, except for the Griffin and the Kahn C scores (for sensitivity) and the Balkau score
55 (for specificity). The numbers needed to screen ranged from 15.5 for the Kahn CB score to 36.7 for the Griffin
56 score.

57 **Conclusion:** The Kahn (CB) and the Findrisc performed best of all scores. Findrisc could be used in an
58 epidemiological setting, while the need of blood sampling for the Kahn (CB) score restricts its use to a more
59 clinical setting.

60 **PRÉCIS**

61 We tested 7 risk scores regarding their ability to predict incident type 2 diabetes after a 10-year follow-up in a

62 Swiss population cohort. Kahn clinical-biological and Findrisc scores performed best.

63 INTRODUCTION

64 The prevalence of type 2 diabetes mellitus (T2DM) is increasing worldwide (1). In Switzerland, one out
65 of sixteen persons aged between 35 and 75 years has diabetes mellitus, and almost one third of diabetic subjects
66 is unaware of their status (2) despite easy access to screening in local pharmacies and general practitioners.
67 T2DM carries a considerable economic burden (3) as patients with T2DM are at higher risk of developing
68 cardiovascular, neurological, renal and ophthalmic complications. Hence, early diagnosis of T2DM is of major
69 importance as the outcome of the disease can be modified through medical care and lifestyle changes (4). The
70 identification of subjects at high risk of developing T2DM might also be cost-effective by reducing the incidence
71 of T2DM (5). Therefore, multiple predictive risk scores have been developed to detect patients at high risk of
72 developing T2DM(6). A review conducted in 2011 (7) identified as many as 145 diabetes risk models or scores
73 and suggested a monthly increase of this number. Such scores rely mainly on anamnestic and clinical information
74 such as personal or family history and on simple measurements such as blood pressure, weight or waist. Some
75 scores use additional blood markers such as fasting glucose, cholesterol and triglycerides. Among the 94 risk
76 prediction models studied by Noble et al, 40 were based on biological variables. (7) While scores including blood
77 markers tend to perform better, their cost is higher (6). Further, most scores have been validated in selected
78 populations, and their application in other settings or populations is not warranted. Health professionals need
79 to be able to rely on robust scores to easily identify the people at risk of diabetes and prevent exaggerated and
80 costly screening in those less at risk.

81 In a previous study, we assessed the 5.5-year predictive capacity of seven T2DM risk scores in a
82 prospective, population-based sample (8). As many scores were originally developed using longer follow-up
83 times, a further validation was deemed necessary. Hence, in this study, we aimed to validate the seven above-
84 mentioned T2DM risk scores over a 10-year follow-up. Our initial hypothesis was that the predictive capacity of
85 each score would not change significantly in a longer follow-up.

86 METHODS

87 *The Colaus study*

88 The sampling procedure of the CoLaus cohort has been described previously (9) and further details can
89 be obtained under www.colaus-psycholaus.ch. Briefly, the source population was defined as all subjects aged

90 between 35 and 75 years registered in the population register of the city of Lausanne. The register includes all
91 subjects living in this city for more than 90 days. A simple, non-stratified random sample of 19'830 subjects
92 (corresponding to 35% of the source population) was drawn and the selected subjects were invited to participate
93 by letter. If no answer was obtained, a second letter was sent, and if still no answer was obtained, the subjects
94 were contacted by phone. Recruitment began in June 2003 and ended in May 2006, enrolling 6733 total
95 participants who underwent an interview, a physical exam, and a blood analysis. The first follow-up was
96 performed between April 2009 and September 2012, 5.6 years on average (median 5.4 years, range 4.5-8.8)
97 after the collection of baseline data; the second follow-up was performed between May 2014 and April 2017,
98 10.9 years on average (median 10.7, range 8.8-13.6) after the collection of baseline data. The information
99 collected was similar to that collected in the baseline examination.

100 *Data collection*

101 Participants were asked to attend an outpatient clinic at the Centre Hospitalier Universitaire Vaudois in
102 the morning, after an overnight fast. History of disease (personal and familial), socio-demographic and lifestyle
103 data were collected by questionnaire. Smoking was categorized as never, former and current; alcohol
104 consumption was assessed by the number of alcoholic drinks (i.e. glasses of wine, cans of beer or shots of spirit)
105 consumed over the last seven days and categorized into none, moderate (1-13 units/week), high (14-27
106 units/week) and very high (28+ units/week). Educational level was categorized as low (primary), middle
107 (apprenticeship), upper middle (high school), and high (university) for highest completed level of education.
108 Physical activity was defined by exercising at least twice a week for at least 20 minutes per session. Prescribed
109 and over-the-counter medicines were collected by questionnaire.

110 Body weight and height were measured with participants barefoot and in light indoor clothes. Body
111 weight was measured in kilograms to the nearest 100 g using a Seca® scale (Hamburg, Germany). Height was
112 measured to the nearest 5 mm using a Seca® (Hamburg, Germany) height gauge. Waist circumference was
113 measured mid-way between the lowest rib and the iliac crest using a non-stretchable tape and the average of
114 two measurements was taken. Blood pressure (BP) and resting heart rate were measured thrice using an
115 Omron® HEM-907 automated oscillometric sphygmomanometer after at least a 10-minute rest in a seated
116 position. Different sized cuffs were available to take into account arm circumference and the average of the last
117 two measurements was used.

118 Venous blood samples (50 mL) were drawn in the fasting state. Biological assays were performed at the
119 clinical laboratory of the Lausanne university hospital within 2 hours of blood collection. Glucose was assessed
120 by glucose dehydrogenase with a maximum inter- and intra-assay CV of 2.1% and 1.0%, respectively; high-
121 density lipoprotein (HDL) cholesterol by CHOD-PAP + PEG + cyclodextrin (3.6%-0.9%); triglycerides by GPO-PAP
122 (2.9%-1.5%), and uric acid by uricase-PAP (1.0%-0.5%). Glycated hemoglobin was measured by high performance
123 liquid chromatography (HPCL) using Bio-Rad, D-10™ system, with measurement range 3.8% (at 18 mmol/mol)
124 to 18.5% (at 179 mmol/mol).

125 *Diabetes risk scores*

126 Seven T2DM risk scores were considered: 1) the FINDRISC (10) ; 2) the Swiss Diabetes Association (SDAS)
127 (11); 3) the clinical and clinico-biological scores by Kahn et al., respectively (12) ; 4) the clinico-biological risk
128 score by Wilson et al. (13); 5) the clinical risk score by Balkau et al. (14), and 6) the clinical risk score by Griffin et
129 al. (15). The details of each score are summarized in **supplementary table 1** (16).

130 The FINDRISC score was derived from the 10-year follow-up FINRISK study consisting of 4435
131 participants (10) and consists of seven variables. The SDAS risk score is adapted from the FINDRISC, using familial
132 history of diabetes as an additional variable. The risk scores by Kahn et al. (12) were derived in a cohort of 15'792
133 adults followed up during 10 years. The clinical risk score (C) consists of nine variables, while the clinic-biological
134 risk score (CB) has four additional biological markers (glucose, triglycerides, high density lipoprotein, uric acid).
135 The clinic-biological risk score of Wilson et al. (13) was derived from the Framingham Offspring Study, where
136 3140 participants were followed up for 8 years; the score consists of six variables among which three biological
137 ones: glucose, triglycerides, and high density lipoprotein. The clinical risk score of Balkau et al. (14) was derived
138 from the DESIR cohort, where 3817 participants were followed for nine years; it consists of four variables. Finally,
139 the score of Griffin et al. (15) was derived from a cross-sectional study consisting of 1077 participants, and is
140 composed of five clinical variables.

141 The FINDRISC, SDAS, Kahn (C and CB), Wilson and Balkau scores are based on a sum of allocated number
142 of points per variable. For the FINDRISC and SDAS, nutritional variables and familial history of diabetes for
143 second-degree parents were not available in our cohort at baseline; thus, the threshold was reduced by 1 point.
144 The Griffin score uses a regression equation to calculate the probability of developing T2DM. As no threshold

145 had been proposed in the original study, a 37% probability was used to identify high-risk individuals, as proposed
146 elsewhere (12).

147 Regarding ethnicity, the Wilson risk score was developed in a sample of 99% white and non-Hispanic
148 subjects, and the Kahn scores were developed in a sample comprising 22% of black people, while no information
149 regarding ethnicity was provided in Findrisc, Griffin and Balkau.

150 *Outcome*

151 The primary outcome was T2DM, defined as fasting blood glucose ≥ 7.0 mmol/l or taking insulin or oral
152 antidiabetic medication as suggested by the American Diabetes association (17). HbA1C was not used in the
153 definition of diabetes, as this variable was only available in the second follow-up.

154 *Exclusion criteria*

155 The original inclusion criteria were: 1) written informed consent; 2) willingness to take part in the
156 examination and to provide blood samples; 3) French language ability. For this study, we added the following
157 exclusion criteria: 1) diabetes (type 1 or 2) at baseline; 2) no follow-up (first or second); 3) missing variables to
158 compute the scores and 4) no outcome data.

159 *Statistical analysis*

160 Statistical analyses were conducted using Stata version 15.1 for Windows (Stata Corp, College Station,
161 Texas, USA). Participants' characteristics were expressed as number (percentage) for categorical variables or as
162 average \pm standard deviation for continuous variables. Between-group comparisons were performed using chi-
163 square or Fisher's exact test for categorical variables and student's t-test or Kruskal-Wallis test for continuous
164 variables.

165 Risk scores were expressed as median and [interquartile range]. The diagnostic capacity of the different
166 risk scores was assessed by the AUC [area under the ROC (receiver operating characteristic) curve] and
167 corresponding 95% confidence intervals (CI). Comparisons of the AUC between scores were performed using
168 the **roccomp** command of Stata. Sensitivity, specificity, positive and negative predictive values and their
169 corresponding 95% CIs were computed using incident T2DM as gold standard. The number needed to screen
170 (NNS) to detect one case of T2DM was computed as the total number of participants screened divided by the

171 number of detected T2DM cases (i.e. true positives). Statistical significance was assessed for a two-sided test
172 with $p < 0.05$.

173 **RESULTS**

174 *Characteristics of participants*

175 Of the initial 6733 participants, 5131 (76.2%) were retained for analysis. The reasons for exclusion are
176 summarized in **figure 1** and the characteristics of the included and excluded participants are summarized in
177 **supplementary table 2** (16). Included participants were younger, had lower waist, body mass index (BMI),
178 prevalence of hypertension and family history of diabetes, and levels of fasting plasma glucose and uric acid
179 than excluded ones. Included participants also had higher caffeine and alcohol consumption and higher levels
180 of physical activity and HDL than excluded ones.

181 **Figure 1:** Exclusion criteria. Results expressed as number of participants and (percentage) using baseline number
182 as denominator.

183

184 *Incidence of T2DM*

185 Upon second follow-up, 405 (7.9%) participants had developed T2DM. The baseline characteristics of
186 the participants who developed T2DM or not are summarized in **table 1**. Participants who developed T2DM were
187 more frequently male, older and former or current smokers, and had higher waist, BMI, prevalence of
188 hypertension and of family history of diabetes, alcohol consumption and levels of fasting plasma glucose,
189 triglycerides, HDL and uric acid, and more frequently received statins.

190 *Performance of risk scores*

191 For each risk score, the median score result and the prevalence of participants at high risk of developing
192 T2DM, overall and according to development or not of T2DM, are provided in **table 2**. The overall prevalence of
193 participants at high risk ranged from 13.7% for the Griffin score to 43.3% for the Balkau score. Prevalence of
194 participants at high risk among those who developed T2DM ranged from 34.6% for the Griffin score to 82.0% for
195 the Kahn CB score (**table 2**).

196 The AUC, sensitivity, specificity, positive and negative predictive values and the number needed to
197 screen to detect one case of T2DM are summarized in **table 3** for each risk score. The AUCs for each score are
198 also provided in **Figure 2** and the results of the bivariate comparisons of the AUCs are provided in **table 4**. The
199 Kahn CB score had the highest AUC while the Griffin score had the lowest. Sensitivities and specificities were
200 above 70%, except for the Griffin and the Kahn C scores (for sensitivity) and the Balkau score (for specificity).
201 Positive predictive values were below 25%, while negative predictive values were above 90%. The numbers
202 needed to screen ranged between 15.5 for the Kahn CB score to 36.7 for the Griffin score (**table 3**).

203 **Figure 2:** ROC curves of the seven diabetes risk scores.

204 **DISCUSSION**

205 Out of the seven diabetes risk scores evaluated, the two with the highest AUC were the Kahn et al. (CB),
206 which includes biological variables, and the Findrisc, which is based on clinical data only. This finding is
207 comparable to what was reported previously using a shorter follow-up period (5.5 vs. 10.9 years) (7).
208 Importantly, our results confirm our hypothesis that the predictive value of a diabetes risk score does not change
209 markedly when shorter follow-up times than the ones in the original validation are used.

210 *Diabetes risk scores*

211 The Kahn (CB) score showed the best metrics, a finding already reported previously (8). Several reasons
212 might explain this performance: first, it was developed using a large sample size (12'729) and included four blood
213 markers. Although the inclusion of blood markers might improve the predictive capacity of the score, it also
214 makes it more expensive to use either for mass screening or for everyday clinical use. Hence, its applicability in
215 settings with limited health resources is reduced. The Wilson score also includes biological markers but, contrary
216 to the Kahn CB, its predictive capacity was rather low and its AUC was comparable to the clinical version of the
217 Kahn score. One explanation could be that Kahn adapts the intervals of seven of its parameters according to sex,
218 whereas Wilson only adapts HDL-C levels. The weighting of each parameter is also different and Wilson was
219 developed on a cohort four times smaller than Kahn.

220 The Findrisc score ranked second highest among all scores. Contrary to the Kahn (CB) score, it is based
221 solely on clinical data and can thus be applied in screening campaigns or in communities with limited health
222 resources. Importantly, although the complete version of the Findrisc score could not be used in this study, still,

223 the reduced version performed well, suggesting that the performance of the complete version, if computable,
224 could even be better. Further, the SDAS, which is based on the Findrisc, also showed an adequate performance,
225 albeit with a lower AUC than the Findrisc. The likely reason is an arbitrary addition of 5 points for family history
226 of diabetes on the SDAS, which does not seem to improve its performance. The Balkau score had the lowest
227 number of components. Although this small number of items might facilitate its applicability in public health or
228 in clinical practice, its predictive capacity was modest, and it led to a very high number of participants classified
229 as being “at risk”. Finally, the Griffin score had the lowest prediction capacity. A probable explanation is that it
230 was developed in a cross-sectional setting, whereas the other scores were developed in a prospective setting.
231 Overall, our results indicate that a prospective setting is paramount to adequately derive and validate a risk
232 prediction score. Indeed, most scores perform well in the populations they were developed in, but their
233 predictive value drops when applied to a different cohort. Hence, externally validating predictive scores on
234 different populations is essential to assess their generalizability and performance.

235 *Comparison with previous findings*

236 Our initial hypothesis was that the predictive capacity of each score would not change significantly in a
237 longer follow-up. The ranking of the scores according to performance is the same for 5- or 10-year follow-ups
238 for the best 3 performers (Kahn CB, Findrisc, Swiss Diabetes association) (**Supplementary Table 3**)(16). Overall,
239 the AUC are slightly lower using 10-year follow-up. Follow-up period transportability is a well-known challenge,
240 as it demands that scores maintain accuracy when predictions are tested over longer versus shorter follow-up
241 periods.(18) In our study, AUC changes are probably due to time-sensitive variables such as anthropometric
242 data.

243

244 *Strengths and limitations*

245 The main strength of our study is that we used a follow-up time similar to the one the scores were
246 developed for. The second strength is the relatively large sample size, which provided an adequate number of
247 incident events.

248 We also acknowledge several limitations. No nutritional data was collected at baseline; hence, we had
249 to adapt the threshold of the Findrisc and SDAS scores by reducing the threshold by one unit, a procedure also

250 performed in our previous study (8). Still, despite this limitation, both the Findrisc and the SDAS scores
251 performed better than other scores. The CoLaus study includes a majority (90%) of Caucasian subjects living in
252 a high-income country and urban setting, who volunteered to participate in the study; hence, full
253 representativeness and generalizability to other ethnicities or settings might not be warranted. Still, no
254 differences were found between the CoLaus cohort and the target population regarding gender and zip code
255 distribution, while subjects aged <65 were underrepresented. Also, it would be important to replicate our study
256 in other cohorts to validate the robustness of our findings. Approximately 15% of our baseline cohort was lost
257 during the first and second follow-up, which might have reduced the number of incident T2DM events. Still, this
258 affected all scores equally and would not change the conclusions of the study. Finally, statins are diabetogenic
259 (19) and participants with incident DM were more frequently prescribed statins at baseline. In this study, we did
260 not assess the predictive value of statin therapy, as the objective was to validate existing DM risk scores.
261 Nevertheless, statins should be considered as a possible T2DM determinant when developing T2DM risk scores.

262 **CONCLUSION**

263 The Kahn (CB) and the Findrisc performed best of all scores. Findrisc could be used in an epidemiological
264 setting, while the need of blood sampling for the Kahn (CB) score restricts its use to a more clinical setting.

265 **LIST OF ABBREVIATIONS**

266	AUC	Area under the ROC (receiver operating characteristic) curve
267	BMI	Body mass index
268	BP	Blood pressure
269	HDL cholesterol	High-density lipoprotein cholesterol
270	HPCL	High performance liquid chromatography
271	Kahn C	Kahn clinical
272	Kahn CB	Kahn clinic-biological
273	ROC	Receiver operating characteristic
274	SDAS	Swiss diabetes association score
275	T2DM	Type 2 diabetes mellitus

276 **DECLARATIONS**

277 **ETHICS APPROVAL AND CONSENT TO PARTICIPATE**

278 The institutional Ethics Committee of the University of Lausanne, which afterwards became the Ethics
279 Commission of Canton Vaud (www.cer-vd.ch), approved the baseline CoLaus study (reference 16/03, decisions
280 of 13th January and 10th February 2003); the approval was renewed for the first (reference 33/09, decision of
281 23rd February 2009) and the second (reference 26/14, decision of 11th March 2014) follow-up. The study was
282 performed in agreement with the Helsinki declaration and its former amendments, and in accordance with the
283 applicable Swiss legislation. All participants gave their signed informed consent before entering the study.

284 **CONSENT FOR PUBLICATION**

285 Not applicable.

286 **AVAILABILITY OF DATA AND MATERIAL**

287 The datasets analysed during the current study are not publicly available as cohort participants did not
288 consent to this. Non-identifiable individual-level data are available for researchers who seek to answer questions
289 related to health and disease in the context of research projects who meet the criteria for data sharing by
290 research committees. Please follow the instructions at <https://www.colaus-psycolaus.ch/> for information on
291 how to submit an application for gaining access to CoLaus/PsyCoLaus data.

292 **COMPETING INTERESTS**

293 The authors report no conflict of interest.

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299 interpretation, writing of the report, or decision to submit the article for publication.

300 **AUTHORS' CONTRIBUTIONS**

301 VK wrote part of the article and revised it for important intellectual content. JF wrote most of the article.

302 GW revised the article for important intellectual content. PMV had full access to the data and is the guarantor

303 of the study. MM was the initiator of the study and revised the article for important intellectual content.

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351 **FIGURE TITLES AND LEGENDS**

352 **Figure 1:** exclusion criteria. Results expressed as number of participants and (percentage) using baseline number
353 as denominator.

354 **Figure 2:** ROC curves of the seven diabetes risk scores.

355

357 **Table 1:** Factors associated with incident diabetes, 10.9-year follow-up, CoLaus study, Lausanne, Switzerland

	No diabetes	Incident diabetes	P-value
N	4726	405	
Gender (women)	2669 (56.5)	149 (36.8)	<0.001
Age (years)	51.3 ± 10.5	55.9 ± 9.8	<0.001
Clinical data			
Weight (kg)	71.3 ± 13.9	82.5 ± 14.2	<0.001
Body mass index (kg/m ²)	25.1 ± 4.0	28.7 ± 4.3	<0.001
Waist (cm)	86.7 ± 12.1	98.7 ± 11.5	<0.001
Hypertension (%)	2073 (43.9)	294 (72.6)	<0.001
Resting heart rate (bpm)	67 ± 9	69 ± 10	0.008
Family history of diabetes (%)	980 (20.7)	137 (33.8)	<0.001
High glucose (≥ 6.1 mmol/L)	1339 (28.4)	318 (78.7)	<0.001
Prescribed steroids (%)	20 (0.4)	3 (0.7)	
Prescribed statins (%)	411 (8.7)	70 (17.3)	<0.001
Lifestyle data			
Alcohol consumption (%)			<0.001
None	1254 (26.5)	107 (26.4)	
1-13 UA/week	2733 (57.8)	212 (52.4)	
14-27 UA/week	601 (12.7)	58 (14.3)	
≥28 UA/week	138 (2.9)	28 (6.9)	
Smoking categories (%)			0.001
Never	1987 (42.0)	132 (32.6)	
Former	1519 (32.1)	151 (37.3)	
Current	1220 (25.8)	122 (30.1)	
Caffeinated drinks consumption (%)			0.135
None	289 (6.1)	25 (6.2)	
1-3 u/day	3075 (65.1)	264 (65.2)	
4-6 u/day	1158 (24.5)	89 (22.0)	
>6 u/day	204 (4.3)	27 (6.7)	
Physical activity (%)	2687 (56.9)	181 (44.7)	<0.001
Blood markers			
Fasting plasma glucose (mmol/l)	5.3 ± 0.5	6.0 ± 0.6	<0.001
Triglycerides (mmol/l)	1.27 ± 0.96	1.99 ± 1.99	<0.001
HDL (mmol/l)	1.68 ± 0.44	1.46 ± 0.37	<0.001
Uric acid (μmol/l)	303 ± 81	353 ± 87	<0.001

358 UA, units of alcohol. Results expressed as average ± standard deviation or as number of participants and
359 (percentage). Between-group comparisons performed using student's t-test or Kruskal-Wallis test (§) for
360 continuous variables and chi-square or Fisher's exact test (†) for categorical variables.

361

362

363 **Table 2:** Bivariate analysis of diabetic risk scores, 10-year follow-up (2003-2006 to 2014-2017) CoLaus study,
 364 Lausanne, Switzerland

	All	No diabetes	Incident diabetes
N	5131	4726	405
Griffin et al.			
Score	11 [3 - 35]	10 [3 - 32]	38 [17 - 66]
High risk (%)	702 (13.7)	562 (11.9)	140 (34.6)
Balkau et al.			
Score	2 [1 - 3]	2 [1 - 3]	3 [3 - 4]
High risk (%)	2222 (43.3)	1905 (40.3)	317 (78.3)
Kahn et al (C)			
Score	25 [12 - 40]	23 [12 - 38]	44 [32 - 57]
High risk (%)	1443 (28.1)	1184 (25.1)	259 (64.0)
Wilson et al			
Probability	3 [3 - 4]	3 [3 - 4]	7 [4 - 18]
High risk (%)	1552 (30.3)	1236 (26.2)	316 (78.0)
Swiss Diabetes association			
Score	7 [4 - 12]	7 [3 - 11]	14 [11 - 17]
High risk (%)	1586 (30.9)	1282 (27.1)	304 (75.1)
Findrisc			
Score	6 [3 - 10]	6 [3 - 9]	12 [9 - 14]
High risk (%)	1388 (27.1)	1096 (23.2)	292 (72.1)
Kahn et al (CB)			
Score	19 [9 - 33]	18 [9 - 30]	47 [35 - 56]
High risk (%)	1426 (27.8)	1094 (23.2)	332 (82.0)

365 Results expressed as median [interquartile range] or as number of participants and (percentage). Between-group
 366 (diabetes and non-diabetes) comparisons using Kruskal-Wallis test or chi-square test. All differences are
 367 significant at $p < 0.001$

368

369 **Table 3:** Diagnostic performance of diabetic risk scores, 10-year follow-up (2003-2006 to 2014-2017) CoLaus
 370 study, Lausanne, Switzerland

	AUC	Sensitivity §	Specificity §	Positive predictive value §	Negative predictive value §	Number needed to screen §§
Griffin et al.	0.740 (0.718 - 0.762)	34.6 (29.9 - 39.4)	88.1 (87.2 - 89.0)	19.9 (17.0 - 23.1)	94.0 (93.3 - 94.7)	36.7
Balkau et al.	0.750 (0.728 - 0.771)	78.3 (73.9 - 82.2)	59.7 (58.3 - 61.1)	14.3 (12.8 - 15.8)	97.0 (96.3 - 97.6)	16.2
Kahn et al (C)	0.777 (0.755 - 0.798)	64.0 (59.1 - 68.6)	74.9 (73.7 - 76.2)	17.9 (16.0 - 20.0)	96.0 (95.4 - 96.6)	19.8
Wilson et al.	0.788 (0.765 - 0.811)	78.0 (73.7 - 82.0)	73.8 (72.6 - 75.1)	20.4 (18.4 - 22.5)	97.5 (96.9 - 98.0)	16.2
Swiss Diabetes association	0.807 (0.787 - 0.828)	75.1 (70.6 - 79.2)	72.9 (71.6 - 74.1)	19.2 (17.3 - 21.2)	97.2 (96.5 - 97.7)	16.9
Findrisc	0.818 (0.798 - 0.838)	72.1 (67.5 - 76.4)	76.8 (75.6 - 78.0)	21.0 (18.9 - 23.3)	97.0 (96.4 - 97.5)	17.6
Kahn et al (CB)	0.866 (0.849 - 0.883)	82.0 (77.9 - 85.6)	76.9 (75.6 - 78.0)	23.3 (21.1 - 25.6)	98.0 (97.5 - 98.5)	15.5

371 Results expressed as value (95% confidence interval). § Of high vs. low risk; §§ to detect one diabetic case.

372

373 **Table 4:** Results of the bivariate comparison of the AUCs between diabetes risk scores.

		Model 2					
		Griffin	Kahn C	Kahn CB	Balkau	SDAS	Findrisc
	Wilson	<0.001	0.358	<i><0.001</i>	0.004	<i>0.047</i>	<i>0.002</i>
	Griffin		<i><0.001</i>	<i><0.001</i>	0.319	<i><0.001</i>	<i><0.001</i>
Model 1	Kahn C			<i><0.001</i>	<0.001	<i>0.001</i>	<i><0.001</i>
	Kahn CB				<0.001	<0.001	<0.001
	Balkau					<i><0.001</i>	<i><0.001</i>
	SDAS						<i>0.029</i>

374 Comparisons were performed using the **roccomp** command of Stata. SDAS, Swiss diabetes association score. In
 375 bold, model 1 performs better. In italics, model 2 performs better.