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## What are the influencing factors on hip and knee arthroplasty survival? Prospective cohort study on 63619 arthroplasties

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1 **Original article**

2

3 **What Are The Influencing Factors on Hip&Knee Arthroplasty Survival? Prospective**

4 **cohort study on 63619 arthroplasties**

5

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17

18

19 Investigation performed at IOR, Bologna, Italy

20

21 **Abstract**

22 *Background*

23 Body Mass Index is used by the World Health Organization to classify obesity. While obesity  
24 influences the onset of arthritis and type-2 diabetes, its effect on implant survival is still open  
25 to debate, with conflicting results from clinical and registry studies, as well as meta-analyses.  
26 Other known factors such as gender or diabetes status could ponderate or mask the effect of  
27 BMI on implant survival.

28 *Hypothesis*

29 Our hypothesis was BMI influenced hip and knee arthroplasty survival, when results were  
30 made independent of gender and diabetes status.

31 *Patient and Methods*

32 A registry study was designed on 30733 Total Hip Arthroplasties (THA), 28483 Total Knee  
33 Arthroplasties (TKA), 3754 Uni compartmental Knee Arthroplasties (UKA) and 649 Hinged  
34 Knee arthroplasties (HK), from 01/01/2003 to 31/12/2015. Mean follow-up was 5.5 years.  
35 Diabetes status was added to the model.

36 Each arthroplasty survival was tested for age at implantation, gender, diabetes status, implant  
37 characteristics and specifically BMI, taking into account gender and diabetes status.

38 *Results*

39 Gender had a strong influence on arthroplasty results. Age also influenced arthroplasty  
40 survival, especially aseptic loosening; a young age would lower implant survival. Diabetes  
41 had an influence in hip survival, but its influence on septic loosening in TKA wasn't proven  
42 ( $p=0.065$ ).

43 A mobile liner and/or a cruciate retaining knee were factors increasing the risk of revision.

44 Weight influenced THA survival, especially aseptic loosening, but didn't have a measurable  
45 effect in any other arthroplasty. BMI was not found to influence any arthroplasty survival,  
46 whatever the endpoint, when diabetes and gender were taken into account.

#### 47 *Discussion*

48 Gender, age and diabetes influenced survival of the lower limb arthroplasties, whereas BMI  
49 did not. Only weight did influence THA results and should be used instead of BMI.

#### 50 *Conclusion*

51 Studies on arthroplasty survival should systematically mention gender and diabetes status and  
52 beware of potential group incomparability.

53 *Level of Evidence:* III, cohort study

54 *Keywords:* Arthroplasty survival; registry; hip; knee; BMI; gender; diabetes

55

### 56 **1. Introduction**

57

58 Obesity is a mechanical influencing factor for osteoarthritis[1] and a metabolic factor  
59 increasing prosthetic joint infections [2]. Its influence on hip or knee aseptic loosening also  
60 could be hinted, as obesity would increase the contact stresses on the implants, however  
61 conclusive evidence seems missing. A registry study by Bordini *et al*[4]. found no influence  
62 of Body Mass Index (BMI) on TKA survival. Kerkhoffs[2] and Si *et al*[5] cohort reviews did  
63 not find a correlation, while another registry study by Culliford *et al*. [6] did find a difference,  
64 but using 'revision for any cause' as the end point.

65 As gender was found to also have an influence on arthroplasty survival[7,8], Smith *et al*. [9]  
66 found that the apparent influence of BMI disappeared when taking into account gender.

67 Obesity carries a higher risk for type-2 diabetes[10], and the effect of diabetes itself on  
68 arthroplasty survival, though controversial[11], could bias the potential effects of weight, or

69 BMI, alone. In this regard, two registry studies by Wagner *et al.* on THA[12] and TKA[13]  
70 have concluded of an effect of BMI on implant revision, but the absence of evaluation of the  
71 diabetes status in the BMI cohorts, as well as gender repartition, could have prevented from  
72 distinguishing the distinct effect of diabetes or gender in the results, thus affecting the  
73 authors' conclusions.

74 In knees, different kinds of arthroplasties could see different quantitative effects of BMI on  
75 revision[14]. Other studies focused on the influence of weight[15], not BMI. This different  
76 approach could prevent the ponderation effect of the height-squared denominator in the BMI  
77 formula.

78 BMI or weight also could have a nonlinear effect along the duration of implantation[2].

79 Our hypothesis was BMI influenced hip and knee arthroplasty survival, when results were  
80 made independent to gender and diabetes status.

81

## 82 **2. Patients and Methods**

### 83 2.1 Patients

84 This study was designed as a registry study (Registro Implantologia Protesica Ortopedica,  
85 Bologna, Italy, <https://ripo.cineca.it>).

86 All analyses were based on primary operations in patients resident in the area of Emilia-  
87 Romagna. The analysis was limited to this cohort of patients. All bilateral cases were  
88 excluded, because of a dependency bias[16]. Were also excluded cases where patient's weight  
89 or diabetic condition was unknown and for THA, cemented or hybrid fixation and metal on  
90 metal (MoM) bearings with head size larger than 32mm. Cemented stems were not found in  
91 sufficient numbers. MoM had issues that could bias the results[17].

92 12802 joints were excluded.

93 The estimation of the presence of diabetes follows Pedersen *et al*[18].

94 2.2 Methods

95 30733 Total Hip Arthroplasties (THA), 28483 Total Knee Arthroplasties (TKA), 3754 Uni  
96 compartmental Knee Arthroplasties (UKA) and 649 Hinged Knee arthroplasties (HK) were  
97 included from 01/01/2003 to 31/12/2015. Mean follow-up was 5.5 years.  
98 Each arthroplasty population was described through age groups, gender and diabetes status  
99 and BMI classification groups (Table 1), to look for group comparability.

100

101 2.3 Methods of assessment

102 A survivorship analysis, with revision for any cause, aseptic and septic loosening as  
103 endpoints, was performed for each type of arthroplasty. For THA was also analysed the  
104 influence of the type of femoral neck; for TKA we also included the influence of the type of  
105 liner and the stabilization model.

106 A Cox proportional hazards model was built to look for the influence of different parameters  
107 on survival, age at surgery, gender, body weight and diabetes status.

108 Another Cox model was made, to look only for the influence of body weight on arthroplasty  
109 survival, without the influence of gender or diabetes status. For each gender and every  
110 arthroplasty, four groups were used: diabetic patients with a normal BMI, diabetic patients  
111 with overweight or obesity, non-diabetic patients of normal BMI versus overweight and  
112 obese..

113

114 2.4 Statistical analysis

115 The statistical analysis was performed using SPSS 14.0 for Windows, version 14.0.1 (SPSS  
116 Inc, Chicago, IL, USA).

117 BMI was set as a continuous variable in order to allow using parametric tests and maximizing  
118 the power of the analysis, but ordinal BMI was also used for comparison.

119 The survival curves and log-rank tests were calculated and plotted according to Kaplan-  
120 Meier. Hazard ratios were calculated via a Cox multivariate regression model.

121

122

### 123 **3. Results**

124 Survival analysis

125 Endpoint: revision for any cause

126 With Diabetes status, no difference between the two populations was found ( $p=0.1635$  for  
127 THA;  $p=0.58$  for TKA,  $p=0.7487$  for UKA and  $p=0.4665$  for HK).

128 When the population was divided in 5 BMI groups, only in THA we found a difference close  
129 to significance ( $p=0.0675$ , Fig 1). It was not the case for TKA ( $p=0.65$ ), UKA ( $p=0.4697$ ) or  
130 HK ( $p=0.7683$ ).

131 With gender, a difference was found in favor of females in hips and hinged knees ( $p=0.0003$   
132 for THA,  $p=0.0424$  for HK). For TKA ( $p=0.1062$ ) and UKA ( $p=0.2034$ ) no survival  
133 difference was proven.

134

135 Fixed liners would have a better survival than mobile liners ( $p<0.0001$ ). A significant  
136 difference was also found in favour of PS knees when comparing CR and PS knees, but only  
137 in the log-rank test ( $p=0.0346$ )

138 Endpoint: revision for septic loosening

139 When we looked at the diabetes status, in total hips was found a difference ( $p=0.0221$ );  
140 diabetic patients had a worst survival with regard of septic loosening. It couldn't be proven  
141 for total knees ( $p=0.0665$ , respectively; Fig 2). In unicompartmental ( $p=0.4376$ ) or hinged  
142 ( $p=0.4277$ ) knees no difference was highlighted.

143 When the population was ranked in 5 BMI groups, no difference could be found in either  
144 arthroplasty (THA  $p=0.6717$ , TKA  $p=0.4873$ , UKA  $p=0.9590$ , HK  $p=0.7627$ ). Figure 3  
145 shows the results for TKA.

146 With gender, a significant difference in favour of females was highlighted in hips ( $p=0.0108$ ),  
147 total knees ( $p=0.0001$ ) and hinged knees ( $p=0.0034$ ) but not in UKA ( $p=0.7487$ ).

148 For arthroplasty-specific issues, *i.e.* THA neck modularity ( $p=0.9739$ ), TKA liner mobility  
149 ( $p=0.2929$ ) or stabilization ( $p=0.3748$ ), we did not find differences when septic loosening was  
150 set as endpoint.

151

#### 152 Endpoint: revision for aseptic loosening

153 Diabetes status did not influence survival for aseptic loosening ( $p=0.1785$  for THA,  $p=0.9657$   
154 for TKA,  $p=0.1186$  for UKA,  $p=0.5433$  for HK). BMI also did not influence survival for  
155 aseptic loosening ( $p=0.8095$  for THA,  $p=0.4517$  for TKA,  $p=0.4561$  for UKA,  $p=0.9064$  for  
156 HK). Gender was not a factor of influence for THA ( $p=0.2034$ ), TKA ( $p=0.8752$ ) or HK  
157 ( $p=0.4645$ ), but we found a significant difference in favor of females in UKA ( $p=0.0175$ ).

158

159 In total knees, fixed liners (versus mobile,  $p<0.001$ ) and posterior stabilized (vs cruciate  
160 retaining,  $p=0.0324$ ) total knees had a better survival concerning aseptic loosening.

161

#### 162 Cox model

163 A Cox model testing age, gender, weight and diabetes as factors and with endpoints revision  
164 for any cause, revision for septic loosening or revision for aseptic loosening was built for  
165 THA (Table 2), TKA (Table 3), UKA (Table 4) and HK (Table 5).

166 A hazard ratio with statistical significance was obtained for almost every arthroplasty with  
167 regard to the influence of gender (male/female), between 1.11 in THA [1.08;1.14] and 1.3 in

168 UKA [1.2;1.4] Hinged knee survival was only influenced by gender in septic loosening (4  
169 [1.16;14.9]).

170 Diabetes hazard ratio in hip septic loosening was evaluated at 2.02 [1.02; 3.68]; as the  
171 difference in total knees was only close to the statistical threshold ( $p=0.068$ ), we couldn't  
172 measure the effect of diabetes in total knee septic loosening, showing the same tendency as  
173 the survival analysis (log rank  $p=0.065$ ).

174 A sub-analysis was performed with the same data, but separating males and females; there  
175 was an influence of diabetes on septic loosening in females (in hip and knees, respectively  
176  $p=0.0262$  and  $p=0.0409$ ) but not in males ( $p=0.8123$  in hips,  $p=0.6321$  in total knees).

177 In TKA, mobile liners had a hazard ratio of 1.286 [1.127; 1.466]; a male gender had a hazard  
178 ratio of 1.91 [1.42; 2.56] in septic loosening.

179 In UKA, gender had an influence on the three events, from 1.25 [1.16; 1.35] in septic  
180 loosening to 1.29 [1.19; 1.39] in aseptic loosening. Diabetes did not influence septic  
181 loosening ( $p=0.153$ ), even when selecting only females ( $p=0.999$ ).

182 In hinged knees, only septic loosening were influenced by the factors we investigated. Aside  
183 from the influence of gender already mentioned, age had a hazard ratio of 0.94 [0.9; 0.99].

184 Weight was only found influencing aseptic loosening in hips, with a risk of 1.0016/kg [1.001;  
185 1.003], with a risk of age on the same event of 1.01/year [1.009; 1.011].

186

### 187 *Influence of BMI when groups were made comparable*

188 For each arthroplasty the population was divided in four groups: diabetic males (DM), non-  
189 diabetic males (NM), diabetic females (DF), non-diabetic females (NF).

190 In THA, no statistically significant difference between the groups was found with regard to  
191 the incidence of BMI on revision for any cause (DM  $p=0.6617$ ; NM  $p=0.2165$ ; DF  $p=0.5374$ ;  
192 NF  $p=0.5714$ ), revision for septic loosening (DM  $p=0.9994$ ; NM  $p=0.5889$ ; DF  $p=0.9994$ ; NF

193 p=0.7617) or even revision for aseptic loosening (DM p=0.4215; NM p=0.8533; DF  
194 p=0.9162; NF p=0.8194).

195 In TKA, no difference was found with regard to the incidence of BMI on revision for any  
196 cause (DM p=0.1015; NM p=0.7290; DF p=0.6390; NF p=0.3353), septic loosening (DM  
197 p=0.0636; NM p=0.9443; DF p=0.9030; NF p=0.2176) or aseptic loosening (DM p=0.8740;  
198 NM p=0.6113; DF p=0.8207; NF p=0.4642).

199 In UKA, no statistically significant difference could be highlighted between the groups with  
200 regard to the incidence of BMI on revision for any cause (DM p=0.8322; NM p=0.5035; DF  
201 p=0.0882; NF p=0.0689), revision for septic loosening (DM p=0.9994; NM p=0.1111;  
202 DF=0.4269; NF p=0.4572) or even revision for aseptic loosening (DM p=0.6691; NM  
203 p=0.1514; DF p=0.1023; NF p=0.4304).

#### 204 **4. Discussion**

205

206 While obesity is usually admitted as a factor influencing the onset of arthritis[1] and type 2  
207 diabetes[10], its direct influence on implant survival is still open to debate.

208 No incidence of BMI on revision could be extracted from the model, when the influencing  
209 factors (gender and diabetes status) were equally distributed[19-24]. We could reject our  
210 hypothesis.

211

212 Gender had a strong influence on arthroplasty results. It also had a strong influence on septic  
213 loosening. This result concurred with findings from Culliford *et al.*[6] and the registry  
214 study from Bordini *et al.*[7,8]. The causes for this gender difference are open for debate.

215 While influencing survival in THA, diabetes only had an influence on septic loosening close  
216 to reaching statistical significance in total knees. Diabetic males did not have an increased  
217 risk compared with non-diabetics on implant septic loosening, which was a new finding to our

218 knowledge; while diabetic females indeed have an increased risk of septic loosening. The  
219 cause of this gender difference is also not clear and should be investigated. As our registry  
220 does not include patients re operated but not having their implants revised, these seemingly  
221 disturbing results[25–30] could be explained by a probably more marked influence of diabetes  
222 on healing issues and soft-tissue infection [31] rather than on septic loosening itself, as Yang  
223 *et al.*[32] . The control of the disease also might impact the results [33]  
224 Weight did influence THA survival but no other arthroplasty for any cause of revision.  
225 In knees, soft tissue could allow dissipating at least part of the constraints on the liner [34].  
226 In TKA, a mobile liner and/or a cruciate retaining knee were factors increasing the risk of  
227 revision, a result also found in other registry studies[35,36].  
228 Studies on hips and knees from Wagner *et al.*[12,13] had opposite results compared with our  
229 findings on the effect of BMI. An explanation could be hinted when looking back at Table 1.  
230 There was a different pattern of diabetes status repartition following BMI in the two genders.  
231 Females also represented 65% of morbidly obese hips while only 56% of obese hips.  
232 As we proved gender and diabetes status influenced survival, a different repartition of one of  
233 these parameters between groups could have biased the results, the patient groups being  
234 incomparable. Smith *et al*[9] similarly found no influence of weight when taking into account  
235 gender.  
236 Data from diabetes status or gender repartition between the BMI groups was not available  
237 from these studies.

### 238 Study limits

239 Our study shared the same limits as every registry study, such as the absence of clinical  
240 findings and satisfaction scores. With septic revisions, if implants were not removed, data was  
241 not available in the registry, so infections treated with only debridement and lavage were not  
242 included.

243 The rule in the case of a Debridement, Antibiotics and Implant Retention is however to  
244 change the modular pieces, considered as failures by our study.

245 While our study has a unique focus on diabetes and implant survival, we did not have  
246 indications on the disease itself.

247

## 248 **5. Conclusions**

249

250 Gender, age and diabetes status influenced survival of the lower limb arthroplasties. BMI did  
251 not have an influence on implant survival. Only weight did influence THA results and should  
252 be used instead of BMI. A follow-up study on hips could allow better comprehend other  
253 potential influencing factors such as head diameter or bearing couple.

254 Studies on arthroplasty survival should systematically mention gender and diabetes status and  
255 their repartition in each group and beware of potential group incomparability.

256 Glycemic marker levels would be interesting in registry studies involving diabetic patients.

257

258 **Conflicts of interest:** Bertrand Boyer and Thomas Neri are consultants for SERF (Décines,  
259 France). Aldo Toni receives royalties from Adler (Milan, Italy). The other authors have no  
260 conflict of interest.

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## 263 **Authors contribution**

264 Susanna Stea and Aldo Toni were responsible for data collect, loss of follow-up reduction  
265 strategies and manuscript overseeing; Barbara Bordini and Dalila Caputo were responsible for  
266 the statistical analysis and survival modeling; Thomas Neri was responsible for manuscript  
267 building and redaction, especially material and methods and results; Bertrand Boyer

268 organized the study, built the manuscript and was responsible for introduction and discussion  
269 parts of the manuscript.

270

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## 8. Tables

Table 1. Repartition of gender and diabetes status according to BMI

BMI	Males		Females	
	Diabetic	Non-Diabetic	Diabetic	Non diabetic
	N (%)	N (%)	N (%)	N (%)
Underweight (<18)	2 (0.1)	74 (0.7)	19 (1.2)	528 (3.1)
Normal (18-25)	304 (21.2)	3276 (30.1)	445 (27.4)	6615 (39.4)
Overweight (25-30)	671 (46.8)	5265 (48.4)	636 (39.1)	6396 (38.1)
Obesity (30-40)	383 (26.7)	1788 (16.4)	414 (25.5)	2354 (14.0)
Morbid Obesity (>40)	69 (0.6)	24 (1.7)	32 (2.0)	141 (0.8)
Missing	51 (3.6)	407 (3.7)	80 (4.9)	759 (4.5)
<b>Total</b>	<b>1435</b>	<b>10879</b>	<b>1626</b>	<b>16793</b>

BMI	Males		Females	
	Diabetic	Non-Diabetic	Diabetic	Non-Diabetic
	N (%)	N (%)	N (%)	N (%)
Underweight (<18)	-	11 (0.1)	7 (0.3)	113 (0.6)
Normal (18-25)	166 (12.7)	1398 (19.7)	308 (12.9)	3866 (21.9)
Overweight (25-30)	592 (45.2)	3708 (52.1)	848 (35.6)	7843 (44.4)
Obesity (30-40)	493 (37.6)	1840 (25.9)	1059 (44.5)	5235 (29.6)
Morbid Obesity (>40)	36 (2.8)	57 (0.8)	117 (4.9)	362 (2.0)
Missing	23 (1.8)	102 (1.4)	42 (1.8)	257 (1.5)
<b>Total</b>	<b>1310 (100.0)</b>	<b>7116 (100.0)</b>	<b>2381 (100.0)</b>	<b>17676 (100.0)</b>

BMI	Males		Females	
	Diabetic	Non-Diabetic	Diabetic	Non-Diabetic
	N (%)	N (%)	N (%)	N (%)
Underweight (<18)	3 (0.3)	0 (-)	2 (0.9)	18 (0.8)
Normal (18-25)	223 (21.2)	15 (9.2)	31 (13.4)	583 (25.3)
Overweight (25-30)	594 (56.4)	85 (52.1)	98 (42.2)	1074 (46.6)
Obesity (30-40)	212 (20.1)	58 (35.6)	87 (37.5)	564 (24.5)
Morbid Obesity (>40)	7 (0.7)	2 (1.2)	9 (3.9)	19 (0.8)
Missing	14 (1.3)	3 (1.8)	5 (2.2)	48 (2.1)
<b>Total</b>	<b>163</b>	<b>1053</b>	<b>232</b>	<b>2306</b>

BMI	Males		Females	
	Diabetic	Non-Diabetic	Diabetic	Non-Diabetic
	N (%)	N (%)	N (%)	N (%)
Underweight (<18)	-	1 (0.7)	-	15 (3.4)
Normal (18-25)	4 (16.7)	41 (30.1)	7 (14.9)	123 (27.8)
Overweight (25-30)	17 (70.8)	68 (50.0)	18 (38.3)	165 (37.3)
Obesity (30-40)	3 (12.5)	21 (15.4)	17 (36.2)	109 (24.7)
Morbid Obesity (>40)	-	4 (2.9)	4 (8.5)	22 (5.0)
Missing	-	1 (0.7)	1 (2.1)	8 (1.8)
<b>Total</b>	<b>24 (100)</b>	<b>136 (100)</b>	<b>47 (100)</b>	<b>442 (100.0)</b>

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416 Table 2. Influence on THA survival

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Parameter	Revision for any cause	Revision for septic loosening	Revision for aseptic loosening
	p	p	p
Age	<0,0001*	0,0035*	<0,0001*
Gender	<0,0001*	0,2246	<0,0001*
Diabetes status	<0,0001*	0,0307*	<0,0001*
Weight	0,0006*	0,1251	<0,0001*

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420 Table 3. Influence on TKA survival.

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Parameter	Revision for any cause	Revision for septic loosening	Revision for aseptic loosening
	p	p	p
Age	<0,0001*	<0,0001*	<0,0001*
Gender	0,4298	<0,0001*	0,6662
Diabetes status	0,5950	0,0682	0,8153
Weight	0,4959	0,5754	0,2822

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424 Table 4. Influence on UKA survival

Parameter	Revision for any cause	Revision for septic loosening	Revision for aseptic loosening
	p	p	p
Age	0,1789	0,0006*	0,0195*
Gender	<0,0001*	<0,0001*	<0,0001*
Diabetes status	0,2137	0,1530	0,3444
Weight	0,9620	0,8331	0,8781

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427 Table 5. Influence on HK survival

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Parameter	Revision for any cause	Revision for septic loosening
	p	p
Age	0,3253	0,0099*
Gender	0,0593	0,0296*
Diabetes status	0,1809	0,2999
Weight	0,1649	0,2390

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Figure 1. Survival analysis according to Kaplan-Meier of the different groups for THA, ranked by Bone Mass Index allowed testing survival differences. Endpoint: revision for any cause (log-rank test,  $p=0.0675$ )

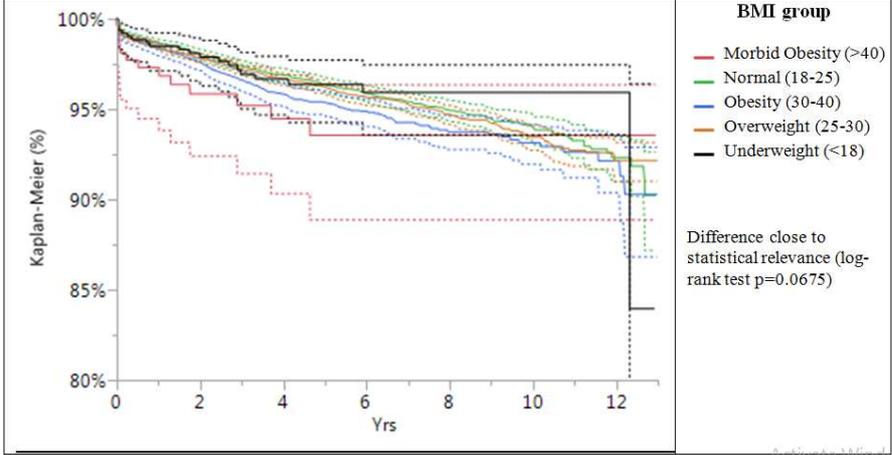


Figure 2. We compared Kaplan Meier's survival curves of diabetic and non-diabetic TKAs, with septic loosening set as end point (log-rank test,  $p=0,0665$ ).

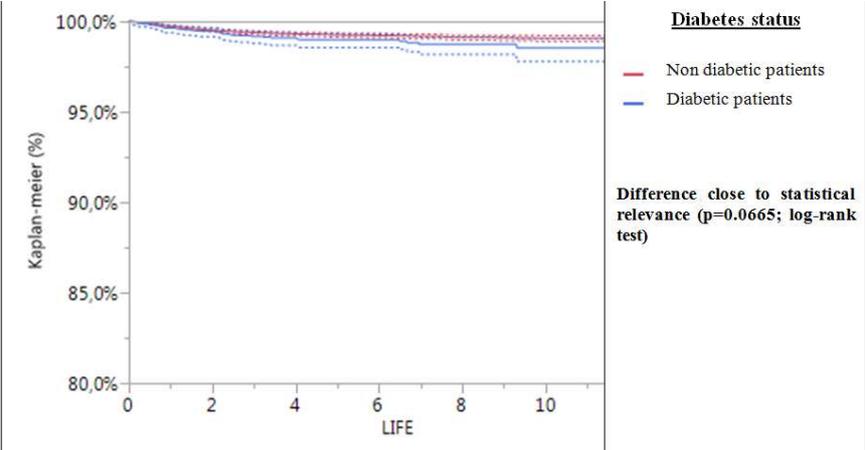


Figure 3. Comparison of Kaplan Meier’s survival curves of TKAs of each BMI group, with septic loosening set as end point (log-rank test, p=0.4873) did not allow highlighting a difference.

