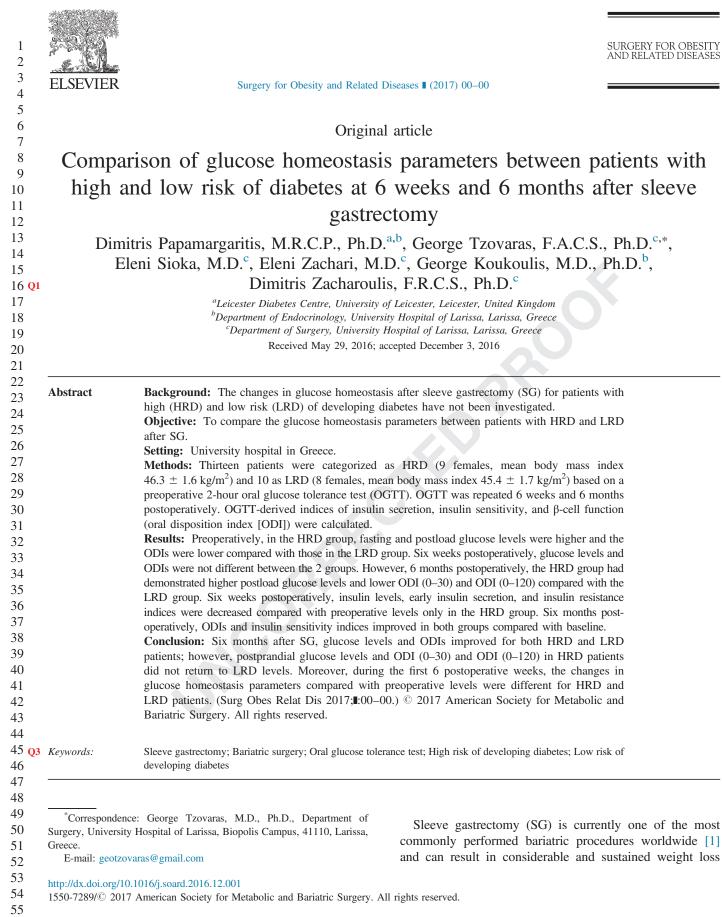
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SURGERY FOR OBESITY AND RELATED DISEASE

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[2,3]. Moreover, SG can restore euglycemia from the early

postoperative period in patients with impaired fasting 64 glycemia (IFG), impaired glucose tolerance (IGT), or type 65 2 diabetes (T2D) [4,5]. Interestingly, studies in patients with 66 T2D have shown that SG also can improve first phase 67 68 insulin secretion and insulin sensitivity immediately postoperatively [6], suggesting that β -cell function may be 69 restored in these patients, at least partially, during the early 70 postoperative period after SG. However, relatively high 71 percentages of diabetes recurrence after SG have been 72 73 reported in the literature [7,8], and a small percentage of 74 patients without diabetes preoperatively (approximately 3%-4%) will develop diabetes within a few years of 75 undergoing SG. These observations suggest that glucose 76 homeostasis is not always fully restored after SG [9]. 77

78 Regarding patients with IFG and IGT, a recent study reported that fasting and 2-hour post-oral glucose tolerance 79 test (OGTT) glucose levels were restored to euglycemic 80 values in all patients 6 months after SG [4]. Nevertheless, 81 whether β -cell function and other parameters of glucose 82 83 homeostasis in patients with high risk of developing diabetes (HRD) are restored to the levels of patients with 84 low risk of developing diabetes (LRD) after SG has not 85 been investigated. 86

The aims of this study were to investigate the changes in 87 88 glucose homeostasis at 6 weeks and 6 months after SG in patients with HRD and LRD and to determine whether 89 glucose levels, β-cell function, and insulin sensitivity in 90 91 patients with HRD are restored to the levels of patients with LRD at these time points. 92

95 Methods 96

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Definition of high and low risk of developing diabetes

99 For this study, HRD patients were categorized as those with fasting glucose levels <126 mg/dL and either 1-hour 100 postload glucose levels ≥ 155 mg/dL or 2-hour postload 101 glucose levels 140-199 mg/dL. On the other hand, LRD 102 103 patients were categorized as those with fasting glucose <100 mg/dL and 1-hour postload glucose <155 mg/dL 104 and 2-hour postload glucose <140 mg/dL. 105 **Q4**

These definitions were based on previous studies that 106 have suggested that patients with IGT (defined as fasting 107 108 glucose <126 mg/dL and 2-hour postload glucose 140-199 mg/dL), patients with normal glucose tolerance (NGT, 109 defined as fasting glucose <100 mg/dL and 2-hour post-110 load glucose <140 mg/dL) and 1-hour postload gluco-111 se \geq 155 mg/dL, and patients with IFG (defined as fasting 112 glucose between 100–125 mg/dL and 2-hour glucose <140 113 mg/dL) and 1-hour postload glucose ≥ 155 mg/dL are at 114 higher risk of developing diabetes compared with patients 115 with NGT and 1-hour postload glucose <155 mg/dL 116 117 [10–12].

Patients with IFG and 1-hour postload glucose <155 118 mg/dL were excluded from the analysis because it is not 119 clear whether they have significantly higher risk of devel-120 oping diabetes compared with patients with NGT and 121 1-hour postload glucose <155 mg/dL [11]. 122

Study participants and design

The study was a retrospective analysis of prospectively 126 collected data. All patients were recruited between March 127 2009 and June 2010 as part of a previous study investigat-128 ing the incidence of dumping syndrome and hypoglycemia 129 after SG [13]. The study was conducted in compliance with 130 the Declaration of Helsinki. Written consent was obtained 131 from each patient, and the institutional review board approved the study.

The inclusion criteria for the study were body mass index 134 $(BMI) \ge 40 \text{ kg/m}^2$ (or $BMI \ge 35 \text{ kg/m}^2$ for patients with 135 obesity related co-morbidities), age ≥ 18 years, and patients 136 without active psychiatric disease. Exclusion criteria for this 137 analysis were uncontrolled drug or alcohol dependency; 138 severely impaired intellectual capacity; previous bariatric 139 surgery; previous diagnosis of diabetes; baseline glycated 140 hemoglobin $\geq 6.5\%$; and the use of oral glucose-lowering 141 medications, glucagon-like peptide 1 receptor analogs, 142 insulin, oral steroids, or beta-blockers. Patients also were 0443 excluded based on their preoperative 2-hour OGTT, 144 whether their fasting glucose was $\geq 126 \text{ mg/dL}$ or the 145 2-hour postload glucose levels were $\geq 200 \text{ mg/dL}$, or 146 whether they were categorized as IFG with 1-hour postload 147 glucose < 155 mg/dL. Overall, 23 patients who fulfilled the Qd 48 inclusion criteria and completed an OGTT 6 weeks and 149 6 months postoperatively were included in the analysis. 150

Patients arrived at the hospital in the morning, after an 151 overnight fast, and an intravenous cannula was inserted. A 152 2-hour OGTT with 75 g of glucose (150 mL of non-153 carbonated glucose drink) was performed. Blood samples 154 were collected at 0 (before oral glucose intake) and at 30, 155 60, 90, and 120 minutes after oral glucose intake to measure 156 glucose and insulin levels. All preoperative OGTTs were 157 performed 4 to 6 weeks before the surgery during the 158 preoperative assessment. 159

The preoperative medications of the patients are pre-160 sented in eTable 1. Patients were asked not to take any T1161 medications on the morning of the preoperative and post-162 operative OGTTs. Blood pressure and cholesterol-lowering 163 medications were stopped during the first months after the 164 SG. Moreover, patients with symptoms of gastroesophageal 165 reflux were advised to take proton pump inhibitors during 166 the postoperative period. 167

All patients underwent SG at a university hospital in 168 Greece. The SG was performed as previously described 169 [14], with dissection starting approximately 5 cm from the 170 pylorus and extending up to the left crus using a 36F bougie 171 to create the gastric sleeve. All patients received the same 172

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