were excluded from the study. All complete blood count analysis was performed by automatic analyser.
Results
We found that mean MPV values and platelet counts in morbid obesity group were higher than control subjects (8.7 ± 1.1 and 6.9 ± 0.5 fl, $P=0.0001$; 267 921.2 ± 81 475/mm^3 0.3; 163 710.8 ± 13 993.3 × 103/μl, $P=0.0001$, respectively). In addition, neutrophil lymphocyte ratio in morbid obesity group was not significantly different than those in control subjects (1.9 ± 0.7 and 1.7 ± 1.0, $P=0.225$, respectively). In addition, platelet to lymphocyte ratio was not statistically different between groups (102.9 ± 32.1 in morbid obesity and 104.8 ± 38.3 in control group). No statistically significant differences were found for the other parameters such as lymphocyte, WBC count and PCT. There were positive correlations both between MPV and BMI ($r=0.649$, $P=0.0001$) and between MPV and body weight ($r=0.599$, $P=0.0001$).
Conclusions
High MPV was associated with the presence of more metabolically active platelets. Therefore, increases of MPV in morbid obesity may lead to high risk for atherosclerosis.

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EP590

Genetic variation in GLP1 receptor is associated with interindividual differences in weight lowering potential of liraglutide in obese women with PCOS

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Objective
The weight lowering potential of glucagon-like peptide (GLP) 1 receptor agonists (RAs) is interindividually different and clinically unpredictable. The potential role of genetic variability of GLP1R on body weight response to GLP1 RA has not yet been evaluated. The aim of the study was to assess the effect of common non-synonymous *GLP1R* single nucleotide polymorphisms (SNPs) rs6923761 and rs10305420 on weight loss in response to long acting GLP1 RA liraglutide in obese women with PCOS.
Methods
59 obese women with PCOS (aged 30.7 ± 6.9, BMI 38.4 ± 5.3 kg/m^2) were assigned to liraglutide 1.2 mg QD sc. for 12 weeks. They were genotyped for *GLP1R* rs6923761 and rs10305420. Changes of body mass, BMI, waist circumference and visceral adipose tissue (VAT) area were measured before and at the end of the study.
Results
After treatment intervention women lost on average 3.96 ± 3.24 kg ($P<0.001$), BMI was reduced for 1.44 ± 1.22 kg/m^2 ($P<0.001$), waist circumference for 3.31 ± 4.13 cm ($P<0.001$) and VAT for 7.05 ± 18.55 cm^2 ($P=0.002$). Twenty (34%) out of 59 subjects were good responders and lost 5% or more of their initial body weight. Carriers of at least one polymorphic rs10305420 allele had worse treatment response compared to carriers of two WT alleles (OR = 0.27, 95% CI = 0.09–0.85, $P=0.025$). Carriers of at least one polymorphic rs6923761 allele tended to have better treatment response compared to carriers of two WT alleles, but the difference was not statistically significant (OR = 3.06, 95% CI = 0.96–9.74, $P=0.058$).
Conclusion
Polymorphism of *GLP1R* rs10305420 accounts for interindividual differences in response to liraglutide regarding weight loss in obese women with PCOS. Future studies will determine whether such genetic variation may be clinically useful in prediction of the weight lowering potential of GLP1 RAs in obese individuals.

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EP591

The efficacy of lifestyle modification in preventing type 2 diabetes mellitus in subjects with impaired glucose homeostasis

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Aims
The aim of our study is to assess the efficacy of lifestyle modification in preventing diabetes mellitus type 2 (DM 2)
Materials and methods
The study included 100 patients (69 m 258 f) 25–65 years with impaired glucose tolerance/impaired fasting glucose (IGT/IFG) and newly diagnosed DM 2. Patients were divided into two groups matched by sex, age, weight, BMI, waist-to-hip ratio (WHR). Research group included 54 patients who received and carried out recommendations of a balanced diet and physical activity. Control group included 46 patients who did not lifestyle modification. The study was 48 weeks. We measured fasting plasma glucose (FPG), 2-h plasma glucose concentrations (2-h PG) following a 75-g oral glucose tolerance test and related to fasting leptin (FL).
Results
Patients of the research group demonstrated reduction of BMI (-2.3 ± 3.1 kg/m^2) and WHR (-0.02 ± 0.025) ($P<0.01$ for all). They had positive dynamics of FPG and 2-h PG concentrations ($P<0.001$). Persons of the control group had increase BMI and WHR as also FPG and 2-h PG concentrations elevation ($P<0.05$). The main novel finding was that median serum leptin in research group decreased on −23.9% ($P<0.01$) and increased in control group on +27.6% ($P<0.01$). Among subjects with IGT from the research group, glucose levels normalised in 49.3% ($P<0.001$) and serum leptin levels decreased on 26.9% ($P<0.01$). In control group glucose levels normalized in 4.5% ($P<0.01$) persons with IGT. By the end of the study 12% of non-diabetic subjects with obese have developed DM 2 and 48% IGT. Among patients of the research group was a reduction of DM 2 by 11.9% and an increase in the control group by 35.1%.
Conclusion
Thereby, lifestyle modifications lead to reduction not only fasting plasma glucose, 2-h plasma glucose concentrations but and fasting leptin concentrations in individuals with impaired glucose tolerance.

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EP592

Relationship between thyroid hormone status and concomitant medication in hyperlipidaemic patients with statin induced adverse effects

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Statins are effective treatment for the prevention of cardiovascular diseases and used extensively worldwide. However, adverse effects induced by statins are the major barrier of maximalising cardiovascular risk reduction. Hypothyroidism and administration of drugs metabolised on the same cytochrome P450 (CYPP450) pathways where statin biotransformation occurs represent a significant risk factor for statin induced adverse effects including myopathy. Simvastatin, atorvastatin and lovastatin are metabolized by CYP3A4, fluvastatin by CYP2C9, while rosuvastatin by CYP2C9 and 2C19.
We investigated the levels of the free thyroid hormones and CYP metabolism of concomitant medication in 101 hyperlipidaemic patients (age 61.3 ± 9.9 years) with statin induced adverse effects including myopathy (56 cases; 55.4%), hepatopathy (39 cases; 38.6%) and gastrointestinal adverse effects (24 cases; 23.8%). Abnormal thyroid hormone levels were found in five patients (4.95%); clinical hypothyroidism in two and hyperthyroidism in three cases. 11patients had a positive history for hypothyroidism (10.9%). There were no significant differences in the TSH, fT_4 and fT_3 levels between patients with myopathy and patients with other adverse effects. 78 patients (77.2%) were administered drugs metabolized by CYP isoforms used by statins (3A4: 66 cases (65.3%); 2C9: 67 cases (66.3%); 2C19: 54 cases (53.5%)). Patients with myopathy took significantly more drugs metabolized by CYP3A4 compared to patients with other adverse effects ($P<0.05$). More myopathy cases were found in patients on simvastatin treatment (52% vs 38%, NS), while significantly less patients with myopathy were on fluvastatin treatment (13% vs 33%, $P<0.05$) compared to patients with other types of statin induced adverse effects. Both abnormal thyroid hormone status and administration of drugs metabolized by CYP3A4, 2C9 and 2C19 are common in our patients with statin induced adverse effects. Normalising the thyroid hormone status and optimising of the concomitant medication may reduce the risk of statin induced adverse effects.

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