Materijali i metode: Svaku skupinu činilo je 30 ispitanika. Katalitičke koncentracije SOD i GPOD određivane su u lizatu eritrocita, a katalaza u plazmi.
Rezultati: Katalitičke koncentracije sva tri enzima statistički značajno su snižene (Grupa A: SOD, P < 0,001; GPOD, P = 0,030; katalaza, P = 0,001; Grupa B: SOD, P < 0,001; GPOD, P = 0,005; katalaza, P < 0,001) u usporedbi sa skupinom zdravih ispitanika što je u skladu s dosada provedenim studijama i potvrđuje kompromitirani antioksidativni status dijabetičara. Vrijednosti katalitičkih koncentracija enzima u dvjema skupinama dijabetičara obuhvaćenih našim ispitrivanjem nisu se statistički značajno razlikovale (SOD, P = 0,451; GPOD, P = 0,375; katalaza, P = 0,546).
Zaključak: Do sada provedene studije različitih autora pokazuju oprečne rezultate što se objašnjava različitim brojem ispitanika, različitim metodama mjerenja ili pak razlikama u dobi pacijenata, tipu i trajanju šećerne bolesti.

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B without DR N = 30) and to compare them with the control group (N = 30).
Materials and methods: Catalitic concentrations of SOD and GPX were determined in RBC lysate and catalase in plasma.
Rezultati: The results for all three enzymes were significantly lower in both diabetic groups (Group A: SOD, P < 0.001; GPX, P = 0.030; catalase, P = 0.001; Group B: SOD, P < 0.001; GPX, P = 0.005; catalase, P < 0.001) compared to the control group, and confirm the compromised antioxidative status of diabetic patients. There was not statistically significant difference of enzymes catalitic activity between the two diabetic group in our study (SOD, P = 0.451; GPX, P = 0.375; catalase, P = 0.546).
Conclusion: Inconsistent results obtained in a number of similar studies could be explained by different number of participants, different methods used, or differences in patients' age, type and duration of diabetes.

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P07 – Koštani metabolizam

P07-1

Status D vitamina u bolesnika na kroničnom liječenju dijalizom i nakon transplantacije bubrega

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Uvod: Poremećaj metabolizma minerala u kroničnom bubrežnom zatajenju posljedica je nedostatne sinteza kalcitriola zbog hiperfosfatemije i smanjenja bubrežnog parenhima. Sinteza 25-OHD u jetri nije poremećena, a nedostatak je posljedica načina života uslijed bolesti, te nakon transplantacije šteti oporavku poremećaja metabolizma minerala.
Cilj ovog rada je procjene statusa D vitamina mjerenjem 25-OHD u 101bolesnika na kroničnom liječenju dijalizom (LD, 53 M, 48 Ž) i u 441 bolesnika s transplantiranim bubregom (TB, 250 M, 191 Ž). **Metode**: Koncentracija 25-OHD određena je ELISA kitom (IDS, Velika Britanija).
Rezultati: U LD bolesnika nije bilo značajne razlike u koncentraciji 25-OHD između spolova, a manjak (< 75 nmol/L) je nađen u 76% (77/101, 35,2 ± 17,7). U 24 bolesnika bez manjka 25-OHD je 118,8 ± 49,4 nmol/L (75-295). Značajno niži 25-OHD u zimskom razdoblju godine (listopad/ožujak) (P < 0,001) 27,9 ± 16,1 (N = 9) nego u ljetnom (travanj/rujan) 57,7 ± 46,7 (N = 92) je relativan zbog malog broja

P07 – Bone metabolism

P07-1

Vitamin D in patients on chronic dialysis treatement and kidney transplant recipients

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Introduction: Disorder of mineral metabolism in chronic kidney failure results from deficient calcitriol synthesis caused by hyperphosphatemia and kidney tissue loss. 25-OHD synthesis in the liver is not affected, its deficiency is a consequence of life style in sickness, and delays normalisation of mineral metabolism after kidney transplantation. The aim of this investigation was assessment of vitamin D status by measurement of 25-OHD in 101 patient on chronic dialysis (CD, 53 M, 48 F) and in 441 kidney transplant recipients (KT, 250 M, 191 F).
Methods: 25-OHD was measured by ELISA kit (IDS, UK).
Results: In CD patients no difference in 25-OHD between sexes existed, and deficiency (< 75 nmol/L) was present in 76% (77/101, 35.2 ± 17.7). In the remaining 24 patients 25-OH D was 118.8 ± 49.4 (75-295). Significantly lower (P < 0.001) 25-OHD in the winter period (October/March; 27.9 ± 16.1, N = 9) than in summer (April/September; 57.7 ± 46.7, N = 92) was considered relative due to small sample. In KT patients 25-OHD was lower in women (45.9 ±

bolesnika. U TB bolesnika 25-OHD je u žena (45,9 ± 26,3; P < 0,005) niži nego u muškaraca (60,7 ± 38,0), ali nema razlike tijekom godine. U 77% (340/441) TB bolesnika postoji manjak 25-OHD, a u 101 iznosi 101,9 ± 31,8 (75-262). Nema razlike između dvije skupina bolesnika niti između spolova za sve. Manjak 25-OHD je u 71% muškaraca (216/304) i 84% (201/239) žena za sve. Manjak D vitamina je nađen u > 75% ovih bolesnika, bez razlike prema osnovnom stanju. U žena je manjak učestaliji i nakon transplantacije su koncentracije niže nego u muškaraca. Doba godine nije utjecalo na 25-OHD.
Zaključak: Veliki udio manjka 25-OHD u žena je nepovoljan je čimbenik nastanka osteoporoze i poremećaja mineralnog metabolizma.

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26.3; P < 0.005) than in men (60.7 ± 38.0), but no difference existed during the year. Deficiency was found in 77% (340/441) KT patients, and in the remaining 101 was 101.9 ± 31.8 (75-262). No difference between sexes was found. In the entire group 25-OHD deficiency was present in 71% men (216/304) and 84% women (201/239). Vitamin D deficiency was found in > 75% patients, without difference regarding diagnosis. In women deficiency was more frequent and after kidney transplantation 25-OHD was less than in men. The seasons did not influence 25-OHD.
Conclusion: Large proportion of 25-OHD deficiency in women is a negative risk factor for osteoporosis and mineral metabolism disorder.

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P08 – Molekularna dijagnostika

P08-1 (Usmeno priopćenje)

Detekcija točkaste mutacije T315I kod bolesnika rezistentnih na terapiju imatinib- mesilatom

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Uvod: Kronična mijeloična leukemija (KML) je mijeloproliferativna bolest karakterizirana nastankom fuzijskog gena BCR-ABL, a posljedično time i fuzijskog proteina bcr-abl. Iako je otkrićem imatinib-mesilata poboljšano liječenje bolesnika s KML, kod manjeg broja bolesnika nakon određenog vremena dolazi do rezistencije na lijek. Jedan od mogućih razloga rezistencije su mutacije u kinaznoj domeni abl. Jedina mutacija rezistentna na danas dostupne tirozin-kinazne inhibitore je točkasta mutacija T315I. Cilj rada bio je detektirati je li prisutnost mutacije T315I uzrok porasta, odnosno visoke razine prijepisa bcr-abl.
Materijali i metode: U istraživanje su bila uključeno 24 bolesnika s KML te neodgovarajućim odgovorom na terapiju imatinib-mesilatom: suboptimalni odgovor na terapiju imalo je 10 bolesnika, 8 bolesnika uopće nije odgovorilo na terapiju, dok je u 6 bolesnika došlo do > 1 log povećanja fuzijskog prijepsa bcr-abl progresivno kroz vrijeme. Napravljen je ASO-PCR prema protokolu Kanga i sur. (Haematologica, 2006; 91:659). Kao pozitivna kontrola, korišten je uzorak DNA bolesnice kod koje je dokazano postojanje mutacije T315I.
Rezultati: Metodom ASO-PCR otkrivena je točkasta mutacija T315I u 4/24 bolesnika (17%). Svi bolesnici kod ko-

P08 – Molecular diagnosis

P08-1 (Oral presentation)

T315I mutation detection in patients resistant to imatinib mesylate therapy

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Introduction: Chronic myeloid leukemia (CML) is a myeloproliferative disease characterized by the presence of BCR-ABL fusion gene and consequently bcr-abl fusion protein. Although the discovery of tyrosine kinase inhibitor (TKI), imatinib mesylate, improved the treatment of CML patients, a proportion of patients develop resistance to drug resulting in increased bcr-abl level. One of possible reasons for resistance are mutations in ABL kinase domain. Some mutations can be overcome by increasing the drug dose or by using the new generation of TKI. The only mutation resistant to currently available TKI is T315I. The aim of this study was to detect if the presence of T315I is the cause for increase or constantly high bcr-abl level.
Materials and methods: Twenty-four CML patients with inadequate response to therapy were included in the study: 10 patients with suboptimal response, 8 patients with failure in response to therapy and 6 patients with > 1 log increase of bcr-abl level over time. ASO-PCR for T315I detection was performed according to Kang et al (Haematologica, 2006; 91:659). Positive control was DNA sample from a patient with confirmed T315I mutation.