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statistically superior TWL when compared to ESG with a statistical p-value of 0.02. EWL and BMI change with LSG and ESG were comparable with a statistical p-value of 0.5 and 0.13 respectively.

Conclusion: ESG, as a minimally invasive treatment modality, demonstrates successful clinical outcomes in patients with obesity, in terms of TWL% EWL%, and BMI. Outcomes at 12 months are similar to LSG, except for TWL that was statistically superior with LSG when compared to ESG. This study was limited by heterogeneity and indirect comparison. Well-conducted large scale studies with adequate follow-up time are needed to establish the role of ESG in the treatment of obese patients.

Disclosure: Nothing to disclose

P0677 PERINATAL PROGRAMMING OF INTESTINAL HOMEOSTASIS FOLLOWING EXPOSURE TO A HIGH FAT DIET IN MALE RATS OFFSPRING

Guibourdenche M.^{1,2}, El Khayat El Sabbouri H.³, Bouzerara A.⁴, Djekkoun N.³, Khorsi-Cauet H.³, Guibourdenche J.⁴, Bach V.³, M Anton P.², Gay-Quéheillard J.³

¹PériTox, Périnatalité & Risques Toxiques, UMR-I 01, UPJV, Amiens, France,

²Equipe PETALES - EA 7519 - Unité Transformations & Agro-Ressources,

UniLaSalle, Beauvais, France, ³PériTox, Périnatalité & Risques Toxiques,

UMR-I 01, UPJV, Amiens, France, ⁴Biologie Hormonale, CHU Cochin,

Université Paris Descartes, AP-HP, Paris, France

Contact E-Mail Address: marion.guibourdenche@outlook.fr

Introduction: Perinatal period is characterized by phases of development with high sensitivity to the environmental factors. Among the risk factors, malnutrition or maternal obesity are now recognized to program children's metabolism and promote the occurrence of obesity or type 2 diabetes during postnatal life. This study aims to identify the effects of maternal perigestational exposure to an obesogenic diet in offsprings. This exposure might increase the occurrence of obesity, associated with metabolic disorders, inflammation and disturbances of digestive function in male offsprings.

Aims & Methods: 8 female Wistar rats were fed a HFD, and 8 control female rats a standard diet (controls), supplemented or not with inulin.

Female rats were exposed to these experimental conditions during a 4-months pre-gestational period as well as during the gestation and lactation periods.

After weaning, 50 male pups were studied at young adulthood (D60), without any treatment during the experiment. Different segments of the digestive tract were studied for histological analysis, metabolic assays, inflammation and intestinal permeability.

Results: Rats from mothers fed a HFD have a higher weight than control pups at weaning time ($p < 0.001$), and the inulin appears to limit this weight gain (HFD vs HFDi $p < 0.05$), phenomenon still present at d60 (C vs HFD $p < 0.01$; HFD vs HFDi $p < 0.01$). Lipid and glycemic assays did not show significant differences. FITC assays didn't show any perturbation of the paracellular intestinal permeability. LPS and pro inflammatory cytokines assays (IL6, IL1 β , TNF α) didn't reveal any tissue inflammation.

Conclusion: Our results indicate that pups from mothers fed an obesogenic diet are overweight at both weaning and young adulthood. Interestingly, inulin limits weight gain in these animals.

The obesogenic diet of the mother promotes the occurrence of obesity in male offsprings and an inulin-based dietary supplement could help limiting these deleterious effects. This hypothesis remains to be confirmed after analysis of the intestinal barrier tight junction proteins expression, other inflammatory markers and morphological alterations of the digestive system.

Disclosure: Nothing to disclose

P0678 ENDOSCOPIC SLEEVE PPLICATION (ESP) FOR TREATMENT OF OBESITY I-II. PRELIMINARY RESULTS OF 2 SITES WITH THE NEW PATTERN FOR GASTRIC EMPTYING DELAY

Turro R.¹, Espinós Pérez J.C.², Lopez Nava G.³, Mata Bilbao A.⁴, Abu Dayyeh B.⁵, Uchima H.⁶, Rosinach M.⁷, Ble Caso M.⁸, Michelena J.⁸, Turro J.⁹

¹Centro Medico Teknon Unidad Endoscopia Digestiva, Endoscopy, Barcelona, Spain, ²Boston Scientific, Endoscopy, Barcelona, Spain, ³Sanchinarro University Hospital, Bariatric Endoscopy Unit, Madrid, Spain, ⁴Centro Medico Teknon, Barcelona, Spain, ⁵Mayo Clinic, Endoscopy, Rochester, United States, ⁶Hospital Germans Trias i Pujol / Teknon Medical Center, Gastroenterology (GI Endoscopy Unit), Barcelona, Spain, ⁷Teknon Medical Center, Endoscopy, Barcelona, Spain, ⁸Teknon Medical Center, Gastroenterology (GI Endoscopy Unit), Barcelona, Spain, ⁹Centro Medico Teknon, Endoscopy, Barcelona, Spain

Contact E-Mail Address: romanturro@gmail.com

Introduction: Obesity is major disease in our society. Intra-gastric balloon is the endoscopic gold standard on short time weight loss. Endoscopic plication can offer us better middle long term results than balloon for its durability.

Aims & Methods: This is a multi-center, prospective pilot study intended to evaluate the safety and efficacy of the Gastric Endoscopic Sleeve Plication procedure (mid & distal body plications) (GESP).

Study was Ethics approved at institutions. Written consent obtained. Indications have been obesity grade II. Use of the Incisionless Operating Platform (IOP)TM (USGI Medical, San Clemente, CA, USA) with a defined new pattern of disposition of the transmural plications with the g-cathTM EZ suture anchors in the greater curvature shortening and tubulizing the stomach to potentially delay gastric emptying and reduce gastric volume / accommodation for an enhanced physiological effect.

Follow up data will be obtained prospectively every 2 weeks initially for the first 2 months and then monthly for the next 10 months on as part of our long term follow-up program that also emphasized changes in unhealthy eating/lifestyle habits.

Gastric emptying studies previously to the intervention, 2 months after and 6 months after intervention are scheduled. Satiety test are also scheduled during the follow up, basal, 2 months and 6 months after the intervention. Liver test with analytics and fibroscan are also done in those patients basal, 2 months and 6 months.

Results: 39 operations in 39 patients were successfully performed (M: 17 F: 22). Mean BMI 36.9 (Range 31.2 - 40.3). Mean number of anchors placed was 18.3. All patients were discharged ≤ 24 hours. No serious adverse events (SAE). % Mean Total body weight loss at 5 months for the 34 patients was 13.93 \pm 4.14 Kg.

Conclusion: The GESP procedure seems to be a safe intervention without significant adverse effects to date. Initial results in weight loss are encouraging. However, long term follow-up and further study remains necessary to assess its value in treating the etiology of obesity.

Disclosure: USGI Consultant Aspire Consultant Allurion Travel Grant

P0679 TRANSLATABLE MODEL OF METABOLIC SYNDROME AND LIVER DISEASE IN SMALL ANIMALS USING PRECLINICAL ULTRASOUND

Babenko L.¹, Bubnov R.^{1,2}, Lazarenko L.M.¹, Spivak M.Y.¹

¹Zabolotny Institute of Microbiology and Virology of NAS of Ukraine,

Department of Problems of Interferon and Immunomodulators, Kyiv, Ukraine,

²Clinical Hospital Pheophania, Diagnostic and Interventional Ultrasound, Kyiv, Ukraine

Contact E-Mail Address: babenkolidiia@gmail.com

Introduction: Diagnostic ultrasound (US) using general US imaging devices can be effectively used for preclinical studies in small animals providing dynamic life-time control [1], furthermore, can enhance drug delivery and therapeutic effects with visual treatment monitoring (theranostic).

Aims & Methods: The aim was to develop model of metabolic syndrome in small animals using general US machines for longitudinal in vivo observation and screening large numbers of cases for facilitating further translation. The modeling of metabolic syndrome performed in compliance with the ethical standards and includes conducting an experiment on laboratory animals (mice, rats, murine) with the introduction of high calorie diet

or industrial fat-enriched diet; and further US monitoring using 5-20 MHz probes of diagnostic US machines in grey scale, Doppler, sonoelastography, M-mode detecting tissue movement, US-guided interventions, injection US contrast agents:

1) for precise diagnosis transabdominal US detecting signs of metabolic syndrome via detailed **imaging of internal organs**: liver size, echogenicity, stiffness, kidneys size, structure, Doppler measuring resistance index (RI) on segmental renal arteries, spleen size, muscle thickness at midfemoral level, assessment of visceral vessels, systemic hemodynamics, etc.;

2) for screening all involved animals we measured the visceral fat thickness (threshold considered as 1.5 mm in mice) on sagittal probe position and collected records of panoramic abdominal scans (in sagittal and transverse probe positions) and measured the largest longitudinal liver size (via sub-costal approach). Weight, body size, laboratory indices (cholesterol, uric acid, glucose, etc.), microbiome, genetic markers were also determined. After sacrificing we evaluated studied organs.

Results: The model was successfully applied to study effects of new drugs: probiotic strains on high calorie-induced obesity model in BALB/c during 21 days [2] and prebiotic effect on high-calorie diet-induced obesity in rats [3]. US detected development metabolic syndrome, endogenous intoxication syndrome, visceral obesity and liver and kidney dysfunction in mouse and rats. Ultrasound data showed visceral obesity, injury of the liver and organs in all experimental animals. We revealed nephropathy signs (thinning, increasing echogenicity of kidney parenchyma, detecting increasing RI in renal arteries (over 0.7) was feasible in rats. Studies using the models demonstrated efficacy of studied strains, substances improving parameters during experiment. All observed changes were confirmed post mortem.

Conclusion: The method of modeling is reliable, allows to monitor metabolic syndrome signs with high translation potential reflecting development disease in humans.

References: 1. Bubnov RV. The use of ultrasound equipment of general use for in vivo study of cerium dioxide nanoparticles introduction in mice. *Ultrason Med Biol.* 2011, 37 (8): 1-S162. <https://doi.org/10.1016/j.ultrasmed-bio.2011.05.750> 2. Bubnov RV, et al. Comparative study of probiotic effects of lactobacillus and bifidobacteria strains on cholesterol levels, liver morphology and the gut microbiota in obese mice. *EPMA J.* 2017;8(4):357-76. <https://doi.org/10.1007/s13167-017-0117-3>. 3. Konopelniuk VV, et al. Efficacy of Fenugreek-based bionanocomposite on renal dysfunction and endogenous intoxication in high-calorie diet-induced obesity rat model-comparative study. *EPMA J.* 2017. <https://doi.org/10.1007/s13167-017-0098-2>

Disclosure: Nothing to disclose

P0680 ASSESSMENT OF METABOLIC SYNDROME IN INFLAMMATORY BOWEL DISEASE REVEALED FTO VARIANT RS9939609 AS A NOVEL GENETIC MARKER OF CROHN'S DISEASE

Dragasevic S.^{1,2}, Stankovic B.³, Kotur N.³, Stojkovic Lalosevic M.¹, Sodic Milutinovic A.^{1,2}, Milovanovic T.^{1,2}, Jovanovic I.^{1,2}, Lukic S.^{1,2}, Milosavljevic T.^{1,2}, Srentic Drazilov S.³, Klaassen K.³, Pavlovic S.³, Muhovic D.⁴, Popovic D.^{1,2}

¹Clinical Center Serbia, Clinic for Gastroenterology and Hepatology, Belgrade, Serbia, ²School of Medicine, University of Belgrade, Belgrade, Serbia, ³Institute of Molecular Genetics and Genetic Engineering, University of Belgrade, Belgrade, Serbia, ⁴Clinical Center of Montenegro, Podgorica, Montenegro

Contact E-Mail Address: dragasevicsanja@gmail.com

Introduction: Metabolic syndrome (MeS) and inflammatory bowel disease (IBD) share common pathophysiological features including chronic inflammation in visceral adipose tissue, but the interplay still remains unrevealed. Great interest has been recently devoted to the association of FTO gene and obesity, although the biological function is still unclear. FTO is a member of a superfamily of Fe (II)- and 2-oxoglutarate-dependent dioxygenases and presents a nucleic acid demethylase. The proposed FTO pathophysiological mechanism includes alterations of methylation-demethylation states of genes expression in metabolically active tissues.

Aims & Methods: Newly diagnosed 94 Crohn's disease (CD) and 98 ulcerative colitis (UC) patients and 91 non-IBD controls with parameters of MeS were analyzed for FTO rs9939609 variant using PCR-ARMS (Polymerase Chain Reaction - Amplification Refractory Mutation System) method.

Results: We analyzed distribution of genetic variant FTO rs9939609, previously associated with obesity, in our study population. The genotype distribution was in Hardy-Weinberg equilibrium in each and total analyzed group. Results showed that FTO AA genotype was more frequent in CD

than UC and control group, 29.8%, 23.5% and 14.3%, respectively. It has been demonstrated that AA genotype was significant predictor of CD occurrence ($p = 0.01$), adjusted for age and gender in the logistic regression model. Compared to TT and TA carriers, carriers of AA genotype had 2.6 higher odds for CD development (OR = 2.6 95% CI [1.2 - 5.4])

Conclusion: The nutrigenetic approach in IBD could improve understanding of obesity-associated complex diseases and contribute to better risk stratification, considering that genetic markers are not influenced by confounding factors such as education, physical activity, social-economic status and diet. Association of FTO variant with CD could direct further nutrigenomic studies in IBD research.

Disclosure: Nothing to disclose

P0681 MICROBIOTA CHANGES INDUCED BY MICROENCAPSULATED SODIUM BUTYRATE

Facchin S.¹, Vitulo N.², Calgaro M.², Buda A.³, Romualdi C.⁴, Perini B.¹, Lorenzon G.¹, D'Incà R.¹, Savarino E.V.¹

¹University of Padua, Department of Surgery, Oncology and Gastroenterology - DiSCOG, Padua, Italy, ²University of Verona, Department of Biotechnology, Verona, Italy, ³UOC Gastroenterology, GI Oncological Surgery, Feltre, Italy, ⁴University of Padua, Department of Biology, Padua, Italy

Contact E-Mail Address: edoardo.savarino@unipd.it

Introduction: Inflammatory bowel disease (IBD) is characterized by severe inflammation of the small bowel and/or the colon leading to recurrent diarrhea and abdominal pain. Butyrate represents one of the final product of saccharolytic fermentation of complex and nondigestible polysaccharides by anaerobic bacteria and has shown anti-inflammatory and regenerative properties, providing symptomatic relief when orally supplemented in patients suffering from a various range of colonic diseases(1). Limited data are available on butyrate effectiveness in patients with IBD due to the difficulties of proving an adequate concentration of butyrate in the colon.

Aims & Methods: We investigate the effect of a microencapsulated form of sodium Butyrate (MSB, Butyrose[®], SILA, Noale, Italy) on the faecal microbiota of patients with IBD. In this prospective-randomized-placebo-controlled study, 49 IBD patients, 19 CD and 30 UC with mild-to-moderate clinical activity were enrolled. Eighteen volunteers were recruited to provide a healthy microbiota model of the local people. Patients with extensive surgery were excluded. After stratification by clinical assessment, colonoscopy, and fecal calprotectin (FC) levels, the patients were randomized to oral administration of MSB (1800 mg/die) or placebo for 2 months, in addition to conventional therapy. Clinical activity was defined according to HBI in case of Crohn's Disease (CD) and Mayo score in case of ulcerative colitis (UC). Before (T0) and after (T1) butyrate treatment, stool samples were collected for faecal microbiota assessment analysis by 16S ribosomal RNA Illumina MiSeq sequencing. Patients completed the quality of life questionnaire in IBD (IBDQ) on T=0 and T=1

Results: We confirmed the evidence of a significant difference ($p < 0.001$) between the microbiota of healthy controls and IBD patients. MSB induced similar changes in the microbiota of IBD patients by increasing the bacteria able to produce shortchain fatty acids (SCFA). However, an increased abundance of butyrogenic colonic bacteria (including genera *Butyrivibrio* and *Subdoligranulum*) were observed in CD patients, whereas in UC patients we observed a major increase of *Lachnospiraceae* (sPLSDA analysis). Clinically, when only patients with calprotectin levels above 250ug/g for CD and 150ug/g(2) for UC were considered, a 30% decrease of calprotectin levels were observed in 67% of CD patients treated with MSB versus 33.3% in those treated with placebo. Subjective improvement in QoL based on IBDQ was significantly observed either in the treatment ($p=0.0046$) and in placebo ($p=0.039$) group. However, a greater effect was observed among the UC patients.

Conclusion: Microencapsulated sodium butyrate supplementation showed an increase of butyrogenic and SCFA bacteria stimulating growth with a mimicking prebiotic effect increasing the production of endogenous and physiological SCFAs with a marked improvement of QoL and reduction of the level of inflammatory markers.

References: [1] Duncan L.P., et al., (2004), Restricted distribution of the butyrate kinase pathway among butyrate-producing bacteria from the human colon., *J Bacteriol*, 2099-106 [2] Lin JF, et al., (2014), Metaanalysis: fecal calprotectin for assessment of inflammatory bowel disease activity, *Inflamm Bowel Dis*, 1407-15

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Arnspang E.; P0448
 Aro P.; P1960
 Arola J.; P0745, P1446, P1524
 Arondel Y.; OP246
 Arora P.; P0538
 Arora V.; OP149, P1102
 Arotcarena R.; OP246
 Arpurt J.P.; P0548
 Arranz L.; P0400
 Arreola V.; OP017, P1956
 Arribas-Anta J.; OP185, P0872, P0873
 Arrivi G.; P1326
 Arroja B.; OP251, P0034, P0230
 Arroyo A.; P1201
 Arroyo M.; P0395
 Arroyo-Villarino M.T.; P1766
 Arruda J.; P0634
 Arshad A.; P0981
 Arstikyte J.; OP316
 Arthur L.; P1212
 Arthursson V.; P0497
 Arulrajan S.; P2069
 Arvanitakis M.; P1487
 Asada M.; P0136
 Asadzadeh Aghdaei H.; **P1670**
 Asadzadeh-Aghdaei H.; P1209
 Asahina Y.; OP076, P1798
 Asai S.; OP296
 Asai Y.; OP222, P0932
 Asaoka D.; OP081, P0169, P1324
 Asaturi A.; P1597, P1611, P1612, P1613
 Asayama N.; **P0148**, P1164
 Ascanelli S.; P1892
 Aschenbrenner E.; P0073
 Asenova B.; OP343, P1758
 Asensio T.; OP090, P0581
 Asensio Antón J.; P1220
 Asfari M.M.; P0520
 Ash N.; P1150
 Ashida M.; P0616, P0826
 Ashida R.; P0133, P0136
 Ashihara N.; **P0095**, **P0818**
 Ashikari K.; P1178, P1198, P1347
 Asido S.; P0539
 Aslam R.; **P1618**
 Asokkumar R.; OP172, OP243, OP264, P0030, P0254, **P0676**, P0870, P1148
 Assaf Y.; P1339
 Assem M.; P0735
 Assenat E.; P1888
 Assis D.; P1539
 Astegiano M.; P0350, P1908
 Aston W.; P1680, P1682
 Atabaki-Pasdar N.; OP071
 Atasoy A.; P1268
 Ateeb Z.; P1502
 Atencia C.; P0475
 Aterido A.; P1041
 Athanasiou T.; OP283
 Athanasiou V.; P0371
 Atkinson D.; P0822
 Atreya R.; OP141
 Atrott K.; OP021, P0450, P1016, P1124, P1860
 Atstupens J.; P0644
 Atsushi K.; P1975
 Attalla M.A.A.; **P1465**
 Attardo S.; P1537, P1579, P1991
 Attili F.; OP290
 Attwood S.; OP089
 Attwood S.E.; OP092, P1274
 Aubert P.; P1718
 Aubourg A.; OP168
 Aucello A.; P1241
 Augustin T.; P0987, P2016, P2017
 Aulló C.; P0395
 Auriemma F.; OP351, P0955, P0959, P0973, **P1634**, P1637
 Ausems M.G.E.; OP294, P0132
 Aust G.; P0369
 Auth M.; OP087
 Auzinger C.; P1450
 Auzolle C.; OP209
 Avallone L.; P1336
 Avalos D.; P0347
 Avedano L.; P0334, P1028
 Avila B.; P1449
 Avivar-Valderas Á.; P0411
 Avni Biron I.; P1744
 Avram L.; P1387
 Awadie H.; P1592, P1885
 Axiaris G.; P1187, P2026
 Axon A.; P1340, P2032, P2033
 Ayadi S.; P0040, P0742, P1023, P1077, P1445, P1448, P1564
 Ayari M.; **P0040**, **P0742**, **P1023**, **P1445**, P1448
 Ayeboa-Sallah B.; **P1359**
 Aykut U.E.; P0029
 Aymeric L.; P1839
 Ayoub Y.; P0249
 Ayoubi M.; P1405, P1406
 Ayrizono M.L.S.; OP032, P1009
 Ayub K.; P1618
 Azevedo M.F.C.; P0429
 Azevedo R.; P1334, P1407
 Aziz A.; P1691
 Aziz I.; OP203, P0663, **P1197**
 Azouz M.M.; P1768
 Azpiroz F.; P0668, P1200, P1215, P1256, P1351
 Azukisawa S.; P1923
 Azuma M.; P0614, P0832
 Azuma Y.; P0848
 Azumi M.; **P0142**, P1666, P1995
 Azzaroli F.; P1418
 Azzopardi N.; P1498, P1643
 Azzouz M.; P1614
 Azzouz M.M.; P0864, P1738, P1740

B

Bâldea V.; P0757
 Bañales J.M.; P0019
 Baatenburg de Jong R.; P0610
 Baatrup G.; P0448
 Baba E.; P1236
 Baba T.; OP182, OP341, P0217, P0900, P0912
 Baba Y.; P2010
 Babayan A.; P0384
 Babenko L.; **P0679**
 Babiichuk Y.; P1736
 Bacchi Reggiani M.L.; P1418
 Bacelo Ruano I.; P0689, P1222
 Bach K.; P0505
 Bach V.; P0677, P0682
 Bachetti F.; **P1882**
 Bachmann J.; OP329, P1521
 Bachmann P.; P1888
 Backes Y.; P0494, P1152, P1875
 Backu E.; **P0073**
 Baconnier M.; P0548
 Baccus P.; P0280, P1034
 Badalamenti S.; P1470
 Badía M.; P0627, P0630
 Badra G.; P0031
 Badrulhisham F.; **P0028**
 Bae J.Y.; P0149, P0150
 Baebler K.; OP021, **P1016**, P1124
 Baek K.; P0422
 Baert F.; OP140, P1109, P1130, P1137, P1916
 Bafutto M.; P0429, P1908
 Baggus E.; **P0660**, **P0661**
 Baghbanian M.; P1404
 Bagnoli S.; P0349
 Bahí A.; OP027, P0806, P1169
 Bai J.; P2053, P2054
 Bai J.C.; P0667
 Baiano Svizzero G.; P1774
 Baidoo L.; OP006
 Baierl A.; P1853
 Baile Maxía S.; **P0223**, P1243
 Bailey A.; P0093, P0524
 Baines J.; OP227
 Baiocchi D.; P1887
 Baiocchi L.; P1241
 Bajador-Andreu E.; OP286
 Bajbouj K.; P0070
 Bajer L.; **OP336**, P0825, **P1451**
 Bajor J.; OP302, P1417, P1479, P1485, P1493, P1810, P2052
 Baka O.; **P1403**
 Bakaeva Z.V.; P1255
 Bakalarz D.; P0562, P0596, P1925
 Baker C.; P1313
 Baker T.; P1089
 Bakheet N.; **P1309**
 Bakkaloglu O.; **P1438**, P1757, P1812
 Bakó K.; P1526
 Bakucz T.; P0936
 Bakula D.; P1252
 Bakulin I.; **P0376**, P2056
 Bal K.; P1961
 Balaban D.V.; **P1651**
 Balaguer F.; OP368, P1180, P1854
 Balan G.; P1627
 Balaskó M.; P0110, P1417
 Balbinot R.A.; P1428
 Balbinot R.S.; P1428
 Balbinot S.S.; P1428
 Baldacci M.P.; P0595
 Baldaia C.; P0356, P0415
 Baldaque-Silva F.; OP152, OP237
 Baldassarre G.; P1507, P1751, P1912
 Baldeon M.; OP056
 Baldi F.; P1887
 Baldissarro I.; P1750
 Baldo V.; P1177
 Baldoni F.; P0089
 Balduzzi A.; **P0810**
 Balensiefer J.I.; P1428
 Balint A.; P1034, P1050, P1624, P1783, P1810
 Bálint E.R.; P0785
 Ballester R.; P0247, P0662, P0924
 Ballester Ferre M.P.; P1781
 Ballet L.; OP015
 Balmana J.; P1180
 Balogh F.; P1783
 Baloglu E.; P1708
 Bamba S.; OP131, P0920, P1631
 Bamias G.; OP146, P0371, P1127
 Ban H.; **P1320**
 Ban L.; P1610
 Bana S.; P1592, P1885
 Bana e Costa T.; P0091
 Banach M.; P1103
 Banai Eran H.; P1744
 Banales J.M.; OP254
 Bañales J.M.; P1041, P1390
 Bancila I.; **P1947**
 Bandhari B.R.; P1102
 Banerjea A.; P1678
 Banerjee R.; OP071, OP255, P1729
 Banescu C.; P0641
 Bang J.Y.; **OP154**, **OP278**
 Bangma A.; **P1043**
 Banhudo A.; P1334, P1407
 Baniya R.; P0144
 Banka N.H.; P1650, P2030
 Banks M.; OP002, P1530, P1951, P1983, P1985
 Bannaga A.; **P0078**
 Bannova N.; P0480
 Bánovčin P.; P1186
 Banovcin P.; OP230, P0137, **P1286**, P1553
 Bansi D.; P0996
 Baquerizo-Burgos J.; OP031, P0111, P0253, P0477, P0546, P0929
 Barange K.; P1628
 Baranova N.; P1833
 Barash Y.; P1051
 Barauskas G.; P0626
 Barba Orozco E.; P1256
 Barbara C.; P0642, P0728
 Barbara G.; OP211, P0504, P1910, P1914
 Barbaro F.; OP050, OP077, P1600
 Barbaros U.; P1268
 Barber Caselles C.; P0668
 Barberio B.; P0576, P0582, P0583, P1275
 Barbieri P.; OP256
 Barbieri R.; P1018
 Barbiero G.; OP072
 Barboi O.B.; **P0587**
 Barbu S.; OP286
 Barbulescu A.; **P1931**
 Barbuscio I.; OP290, P1275
 Barca A.; P1887
 Barchi A.; P1507, P1751, P1962
 Bardella M.T.; P1362
 Bardone M.; P0173
 Baretton G.; OP227
 Bargo D.; P0387, P0388
 Barigelletti G.; P1362
 Barišić A.; P1732, P1743, P1752
 Barjas E.; P0201, P0722
 Barker L.; P1563
 Barkov A.; P0696
 Barkun A.; OP298
 Barkun A.N.; P0186, P0887
 Barletti C.; P0973
 Barlow G.; OP054, P1146
 Barna G.; P0473
 Barnes C.; OP138
 Barnich N.; OP209, P0318
 Baron T.H.; OP042
 Barone M.; P0428, P0465
 Barquero D.; P0872, P0873
 Barrabés Vera S.; P1707
 Barrachina M.D.; P0313, P0315, P1006, P1698
 Barragry J.; P2069
 Barrajón Masa A.; P1432
 Barranco L.E.; P0627, P0630
 Barranco Castro D.; **P0739**
 Barreau F.; P0451
 Barreiro P.; P0164, P0219
 Barreiro de Acosta M.; P0407, P1795