Endocrine Abstracts

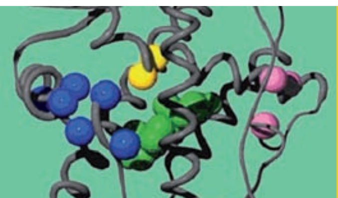
April 2013 Volume 32
ISSN 1479-6848 (online)

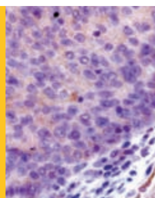
15th European Congress of Endocrinology

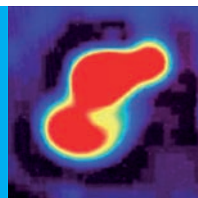
27 April – 1 May 2013, Copehagen, Denmark

ECE2013
27 April - 1 May 2013
Copenhagen, Denmark

European Society of Endocrinology







published by
bioscientifica

Online version available at
www.endocrine-abstracts.org

Endocrine Abstracts

Volume 32
April 2013

15th European Congress of Endocrinology

27 April – 1 May 2013

SPONSORS

The ESE would like to thank the ECE 2013 sponsors

Gold Sponsors:
Ipsen
Novartis
Novo Nordisk
Pfizer

Bronze Sponsors:
Bayer Healthcare
Otsuka

ESE Office
Euro House
22 Apex Court
Woodlands
Bradley Stoke
Bristol BS32 4JT, UK

Contact: Andrea Davis
Tel: +44 (0)1454 642247
Fax: +44 (0)1454 642222
E-mail: info@euro-endo.org
Web site: www.ese-hormones.org

ECE 2013 Secretariat
BioScientifica Ltd
Euro House, 22 Apex Court
Woodlands
Bradley Stoke
Bristol BS32 4JT, UK

Tel: +44 (0)1454 642240
Fax: +44 (0)1454 642222
E-mail: ece2013@endocrinology.org
Website: http://www.ece2013.org

CONTENTS

15th European Congress of Endocrinology 2013

PRIZE LECTURES AND BIOGRAPHICAL NOTES

The European Journal of Endocrinology Prize Lecture EJE1
The Geoffrey Harris Prize Lecture GH1

PLENARY LECTURES

Nutrient-sensing pathways in ageing PL1
NET Management PL2
Changing character of thyroid cancer PL3
Fondation IPSEN 2013 Endocrine Regulations Prize PL4
Preventing vascular complications of diabetes PL5
The Ubiquitin System PL6
Aldosterone, Mineralocorticoid Receptors and Cardiovascular Risk: What's New? PL7
New genes and functions in reproduction PL8
Human Brown Fat is on Fire PL9

SYMPOSIA

Metabolic surgery S1.1–S1.3
Cushing's Disease with negative pituitary imaging S2.1–S2.3
Female reproduction S3.1–S3.3
New advances in GPCRs in endocrinology S4.1–S4.3
A guide through the labyrinth of neuroendocrine tumours S5.1–S5.3
What's new in type 2 diabetes? S6.1–S6.3
Translational aspects from comparative to clinical endocrinology S7.1–S7.3
Action of glucocorticoids on bone S8.1–S8.3
New data treatment of hyperglycemia S9.1–S9.3
Salt-water balance S10.1–S10.3
New mechanisms in SST analogue response S11.1–S11.3
Male reproductive endocrinology S12.1–S12.3
Hormonal treatment in transition of patients with rare diseases (Supported by the *European Journal of Endocrinology*) S13.1–S13.3
Clinical care of the pheochromocytoma patient S14.1–S14.3
The Frail Male S15.1–S15.3
Oncogenic signals in thyroid cancer - therapeutic prospects S16.1–S16.3
Medical treatment of endocrine malignancies - an update S17.1–S17.3
PCOS S18.1–S18.3
Recent advances in the molecular study of endocrine tumours: microRNAs and more S19.1–S19.3
New mechanisms of energy balance S20.1–S20.3
Multi-centre pituitary studies S21.1–S21.3
Improving diagnosis of primary aldosteronism S22.1–S22.3
Endocrine disruptors (Supported by *Endocrine Connections*) S23.1–S23.3
Redefining our understanding of the causes of obesity S24.1–S24.3
Rare metabolic bone disease S25.1–S25.3
Novel technologies and inspiring ideas: From basic endocrine research to clinical practice (European Young Endocrine Scientists (EYES) Symposium) S26.1–S26.3
Steroids in obesity and metabolism S27.1–S27.3
Autoimmune endocrine disease - Old and new players S28.1–S28.3
Management of thyroid nodules S29.1–S29.3

Energy Status and pituitary function S30.1–S30.3
Clinical impact of rare mutations in endocrinology S31.1–S31.3
Is diabetes a lipid disease? S32.1–S32.3

MEET THE EXPERT SESSIONS MTE1–MTE16

JOE/JME PRIZE PRESENTATION *Sponsored by Journal of Molecular Endocrinology*

Enhancing radioiodine uptake in thyroid cancer JP1

ENDOCRINE NURSING SYMPOSIUM EN1.1–EN3.5

ORAL COMMUNICATIONS

Pituitary & Molecular Endocrinology OC1.1–OC1.6
Bone & Calcium OC2.1–OC2.6
Thyroid OC3.1–OC3.6
Adrenal OC4.1–OC4.6
Reproduction OC5.1–OC5.6
Diabetes & Obesity OC6.1–OC6.6

NURSE POSTERS N1–N5

POSTER PRESENTATIONS

Adrenal cortex P1–P64
Adrenal Medulla P65–P69
Bone and Osteoporosis P70–P110
Calcium and Vitamin D metabolism P111–P171.1
Cardiovascular Endocrinology & Lipid Metabolism P172–P212
Clinical case reports - Pituitary/Adrenal P213–P269
Clinical case reports - Thyroid/Others P270–P331
Developmental Endocrinology P332–P345
Diabetes P346–P496
Endocrine disruptors P497–P507
Endocrine tumours and neoplasia P508–P573
Female reproduction P574–P620
Growth hormone IGF axis - basic P621–P636
Male reproduction P637- P677
Neuroendocrinology P678–P718.1
Nuclear receptors and signal transduction P719–P725
Obesity P726–P790
Paediatric endocrinology P791–P822
Pituitary - Basic (*Generously supported by IPSEN*) P823–P839
Pituitary - Clinical (*Generously supported by IPSEN)* P840–P967
Steroid metabolism and action P968–P976
Thyroid (non-cancer) P977–P1076
Thyroid cancer P1077–P1140

INDEX OF AUTHORS

Results
At 1 month evaluation, calcitonin hypersecretion was significantly decreased in seven patients (by 32–73%), stable in six and progressive in one other. Among the ten patients who were evaluable at 3 month follow-up, calcitonin was significantly decreased in six patients, stable in three and further increased in one. Five patients with bone and bowel symptoms at baseline experienced clinical response. Target tumor lesions were stable in 9/10 patients and progressive in 1/10 who had a 3- and 6-month CT scan. FDG-PET SUV_{max} was decreased by 36–50% in 2/9 patients with stable disease at CT scan.
Conclusions
This is the first experience on the use of pasireotide (SOM230) in patients with MTC. An antisecretive and antiproliferative response to pasireotide LAR has been observed in patients with progressive MTC.

DOI: 10.1530/endoabs.32.P1080

P1081

A novel multi-target pyrazolopyrimidine derivative with anti-neoplastic properties, CLM29, is active against medullary thyroid cancer *in vitro* and *in vivo*

Poupak Fallahi, Silvia Martina Ferrari, Guido Bocci, Concettina La Motta, Ilaria Ruffilli, Andrea Di Domenicantonio, Alda Corrado, Caterina Mancusi, Romano Danesi, Federico Da Settimo, Paolo Miccoli &
Alessandro Antonelli
University of Pisa, Pisa, Italy.

Introduction
CLM29, a pyrazolo[3,4-*d*]pyrimidine compound, inhibits several targets (including the RET tyrosine kinase, epidermal growth factor receptor, vascular endothelial growth factor receptor and has an anti-angiogenic effect). Recently it has been shown to inhibit proliferation and migration in primary papillary dedifferentiated thyroid cancer cells. The aim of this study is to evaluate the anti-tumor activity of CLM29 in medullary thyroid cancer (MTC).
Methods/design
The CLM29 anti-proliferative and proapoptotic effects (5, 10, 30, 50 μmol/l) were tested *in vitro* in primary MTC (P-MTC) cells obtained at surgey, in TT cells harboring (C634W) RET mutation, and in human dermal microvascular endothelial cells (HMVEC-d). TT cells were injected in CD nu/nu mice which were treated with CLM29.
Results
CLM29 (10 μmol/l, 30 μmol/l or 50 μmol/l) inhibited significantly ($P<0.001$) the proliferation of P-MTC, or TT cells. CLM29 increased the percentage of apoptotic cells in TT and P-MTC cells dose-dependently ($P<0.001$), while had no effect on migration and invasion. CLM29 inhibited significantly the proliferation, blocking extracellular regulated kinase 1 and 2 phosphorylation and inducing apoptosis in HMVEC-d. The inhibition of proliferation by CLM29 was similar in P-MTC cells with/without *RET* mutation. TT cells were injected s.c. in CD nu/nu mice and tumor masses became detectable between 20 and 30 days after xenotransplantation. CLM29 (50 mg/kg per die) inhibited significantly tumor growth and weight and the therapeutic effect was significant from the 48th day after cell implantation (18 days after the beginning of treatment). A significant reduction of Ki-67 immunostaining and of microvessel density was observed in the CLM29-treated tumors.
Conclusion
The anti-tumor activity of a 'pyrazolo[3,4-*d*]pyrimidine' compound, CLM29, has been shown in MTC *in vitro*, and *in vivo*, opening the way to a future clinical evaluation.

DOI: 10.1530/endoabs.32.P1081

P1082

Association of pre-miR-146a rs2910164 GG genotype with papillary thyroid cancer: a new case–control study on two adjacent genes on chromosome 5, pre-miR-146a and PTTG1.

Marco Marino[1], Valentina Cirello[2], Valentina Gnarini[1], Elisa Pignatti[1], Livio Casarini[1], Chiara Diazzi[1], Vincenzo Rochira[1], Katia Cioni[1], Bruno Madeo[1], Manuela Simoni[1] & Laura Fugazzola[2]
[1]Unit and Chair of Endocrinology and Metabolism, Department of Biomedical, Metabolic and Neural Sciences, University of Modena and Reggio Emilia, Azienda AUSL of Modena-NOCSAE of Baggiovara, Via Giardi, Modena, Italy; [2]Department of Medical Sciences, University of Milan, Endocrine Unit-Padiglione Granelli, Istituto di Ricovero e Cura a Carattere Scientifico Ca' Granda, Via Francesco Sforza, 35, 20122 Milano, Italy.

Introduction
Papillary thyroid carcinoma (PTC) is the most common endocrine malignancy, with a steadily increasing incidence in the last few decades worldwide. Studies revealed the predisposition to PTC by the heterozygous state of rs2910164 within the precursor of microRNA146a. Interestingly, on the same chromosome, 40Kb separate the *pre-miR-146a* from the pituitary tumour transforming gene (*PTTG1*), a proto-oncogene involved in thyroid carcinomas. A genome-wide study revealed an association of the genomic region encompassing *pre-miR-146a* and *PTTG1* gene with systemic lupus erythematosus. In this study, we analyzed, with a case–control design, the genetic association between PTC and *pre-miR-146a* rs2910164 as well as *PTTG1* (rs1862391A/C and rs2910201C/T).
Methods
Two hundred and six healthy controls (30–78 of age) and 307 PTC patients (30–74 of age) were enrolled. The diagnosis of PTC was histological at surgery. Thyroid sonography was performed in controls to exclude nodules. SNP genotyping of pre-miR-146a and PTTG1 was performed by Sanger sequencing and high resolution melting. Linkage disequilibrium (LD) analysis and statistics were performed with Haplowiev 4.2 and GraphPad Prism5 software.
Results and conclusions
Pre-miR-146a rs2910164 allelic frequencies were not statistically different in patients ($C=24.3\%$) and controls ($C=28.6\%$) and the SNP was not in LD with the investigated *PTTG1* SNPs. We did not confirm a previously described association of the CG genotype with PTC. However, a significant association between the GG genotype and PTC (GG vs GC+CC odds ratio=1.38, 95% CI 0.8–2.4) was found. The *PTTG1* SNPs (rs1862391A/C and rs2910201C/T), in perfect LD, have the same allelic frequency in patients ($A=76.7\%$) and controls ($A=76.2\%$) and are not associated with PTC. In conclusion, the study showed a new evidence of association between *pre-miR-146a* rs2910164 and PTC while *PTTG1* did not seem to be involved.

DOI: 10.1530/endoabs.32.P1082

P1083

Circulating microRNAs may help to differentiate malignant from benign thyroid nodules

Tania Pilli, Sandro Cardinale, Silvia Cantara, Giulia Busonero, Francesco D'Angeli & Furio Pacini
Department of Clinical, Surgical and Neurological Sciences, Section of Endocrinology, University of Siena, Siena, Italy.

Introduction
MicroRNAs (miRNAs) are small, endogenous, non-coding RNAs that act as negative regulators of gene expression. The miRNA expression is impaired in many types of human cancer including thyroid cancer. The tissue profile of miRNAs has been shown to be useful for differentiating benign from malignant thyroid nodules, however attainment of tissue samples requires an invasive procedure while blood sampling is minimally invasive and easy to obtain. The aim of this study was to evaluate the circulating levels of a series of miRNAs in 46 patients with nodular goiter in order to identify those that might be useful in the differential diagnosis of thyroid nodules.
Methods
Thirteen miRNAs (miR-222, miR-221, miR-146a, miR-146b, miR-21, miR-155, miR-181a miR-181c, miR-7, miR-30d, miR-126, miR-374th, miR-let7g) were extracted from serum, reverse transcribed, subjected to real-time PCR and then analyzed by the $\Delta\Delta C_t$ method. 10/13 miRNAs were evaluated post-surgically in a subset of patients undergone thyrodectomy.
Results
41/46 patients performed fine-needle aspiration cytology of the dominant nodule (20 benign, three non-diagnostic, six indeterminate, four suspicious for malignancy and eight malignant) and 28/46 patients underwent total thyroid-ectomy (14 benign lesions and 14 papillary thyroid cancer (PTC)). MiR-21 and -222 were higher in patients with benign histology compared to malignant. On the contrary miR-374a was significantly higher in patients with suspicious or malignant cytology and with PTC compared to those with benign disease. After thyroidectomy, the majority of miRNAs decreased while a minority of miRNAs increased or remained unchanged. Moreover miR-7 was significantly lower in patients ablated with radioiodine compared to those treated only surgically.
Conclusions
Our data, although preliminary, suggest the utility of circulating miRNAs (miR-374a showing the best diagnostic accuracy) in the differential diagnosis of thyroid nodules and the lower expression of miR-7 in patients ablated suggests its potential use as a tumor marker.

DOI: 10.1530/endoabs.32.P1083

Author Index

Ágústa Sigurjónsdóttir, H P37
Álvarez, Á P292
Álvarez-Escolá, C P868
Álvarez-Escolà, C P712
Åsman, P P1010
Aşık, M P263, P308 & P309
Abarca, J P908
Abasolo, EU P1127
Abbondanza, C P524
Abd El Baki, R P626
Abdel Aziz, M P626
Abdelbaki, R P385
Abdelrahmanm, O P643
Abdelrazek, S P981
Abdelsalam, M P385
Abderahmane, SA P1039
Abdesselem, H P590 & P949
Abdo, R P507 & P769
Abdoli, S N3
Abdurakhmanova, A P822
Abel, CW P252
Aberle, J P760
Abrams, P N1 & P35
Abreu, A P381
Abreu, C P755
Abreu, S P235
Abrosimov, A P1110 & P832
Abrosimov, AY P1105
Abu-Asab, M P534
Abylayuly, Z P1024
Acar, BC P219
Acconcia, F P720 & P721
Ach, K P611 & P628
Achir, S P1077 & P960
Acibucu, F P120 & P959
Ackermann, C P931
Acs, B P137, P158 & P533
Acs, O P137, P158 & P533
Adam, G P259
Adamczewski, Z P987
Adamek, D P836
Adamidou, F P107, P208 & P92
Adamska, A P296, P346, P351 & P735
Adamska, E P360
Adana, MRd P463, P489 & P490
Adel, C P626
Adina, T P1037
Adolf, C P10
Adorini, L P738
Adrian, D P898
Afanasyev, D P505
Afiane, M P1137
Aflorei, ED P825
Afonso, A P837
Afonso, LP P568
Afzal, N P337
Agapidou, A P107
Agapito, A P837
Agostino Sinisi, A P659, P665 & P678
Aguilar, AH P303
Aguilar-Diosdado, M P474, P493, P56, P745 & P764
Ahluwalia, R P72 & P73
Ahmad, A P441
ahmad, L P441
Ahmadi, J P596
Ahmed Naseem, A P44
Ahmed, A P322
Ahmed, D P496
Ahmed, S P1122
Ahmed-Ali, L P1134 & P1135
Ahmeti, I P253, P427, P559 & P955
Ahn, HY P376, P742 & P762
Aigelsreiter, A P201
Aimaretti, G P467, P473, P851 & P907
Aissa, NB P138, P166 & P167
Ajduk, M P785
Ajdzanovic, V P835 & P838
Ajlouni, K P640
Akaishi, J P536
Akalin, NS P919
Akbal, E P259, P263, P308 & P309
Akbay, E P916
Akcay, S P1023
Akcicek, F P1053 & P506
Akhtar, P P852
Akhtar, S P174
Akin, F P618, P619, P935, P940, P946 & P962
Akin, KO P406
Akin, S P1006, P369, P593 & P62
Akkache, L P212, P237, P571, P621, P63, P933, P945, P957 & P958
Akkurt, A P1086, P1090, P226, P451, P471 & P674
Akman, U P593
Akoz, Z P406
Akram, M P42, P44 & P852
Aksglaede, L OC5.5, P794 & P799
Aksoy, Dy P581
Aktürk, M P1086 & P1090
Aktas, C P446
Akturk, M P1128
Akulevich, N P776
Al Ghuzlan, A P11 & P544
Al Yafei, F P992
AL-Deen, AS P441
AL-Gelany, S P441
Al-Massadi, O P681
Al-Naimi, L P992
AL-Sagheer, G P441
Alagol, F P135
Alali, M P813
Alameda, C P431
Alayev, D P149
Albano, A P21 & P917
Albarel, F P649
Albert, B P898
Albert, K P250
Albert, T P898
Albertelli, M P514
Albertini, S P124
Alberto Gómez, L P777
Alborg, VC P377
Alcántara, V P19
Alcantara, VA P1111
Alecu, M P546
Alecu, S P546
Alevizaki, M P1085, P1121, P187 & PL3
Alexandrescu, D P805
Alexandrou, A P187 & P207
Alexiu, F P801, P805 & P811
Algün, E P257, P277 & P51
Alhumaidi, N P813
Ali Tam, A P1102
Aliev, A P998
Alieva, D P718
Alikhah, H P465
Alimukhamedova, G P718
Alina, S P1037
Alioglu, B P1016
Aller, J P696 & P868
Allo, G P356
Allochis, G P467 & P473
Allolio, B OC4.2, P516 & P702
Almabouada, F OC6.6 & P683
Almanza, MR P377, P564 & P651
Almaraz, MC P133
Almarri, N P992
Almeida, R P890 & P948
Almind, D P984 & P986
Almstrup, K OC5.5 & P666
Alonso Merino, E P719
Alonso-Merino, E P722
Aloumanis, K P815
Altieri, B P23
Altindag, K P62
Altindag, T P386
Altinova, A P1128
Altunrende, B P426
Altuntas, Y P108, P111, P258, P286, P305, P328 & P983
Alvarez Coca, M P714
Alvarez, C P1084 & P880
Alvarez, ED OC3.4
Alves de Santana, A P746
Alves, M P1101, P267, P40 & P563
Alviggi, C P586
Amado, JA P417
Amaral, B P276
Amaral, C P733
Amaral, D P806
Amato, AA P498
Ambekar, S P419
Ambrosi, B P4 & P876
Ambrosio, MR P519, P540 & P907
Ambroziak, U P18 & P592
Amini, M N3
Amini, R P1113
Amirou, AL P810
Amirou, L P1135
Amokrane, L P1137

Rasmussen, LB P1014
Rasolonjanahary, R P334
Rastegar, F P596
Rastrelli, G P655, P657, P658, P668, P672, P675, P676, P677, P680 & P758
Raverot, G OC1.4, P828 & S21.1
Raverot, V OC1.4
Razali, AM P206
Rea, F P519
Recabarren, MP P645
Recabarren, SE P645
Redaelli, M P1099
Redon, J P737
Rees, D P2
Refetoff, S S26.1
Regadera, J P719 & P722
Reghina, AD P413
Rehfeldt, C P757
Reimondo, G P4
Reincke, M OC4.5, P10 & P511
Reinholz, C P342
Reining, F P760
Reis, R P717
Reisch, N P7
Reiter, MH P728
René-Corail, F P5
Rendón-Ramírez, EJ P452
Rendeiro, P OC6.1
Renner, U P833
Rentziou, G P1085 & P1121
Repokis, I P517
Reséndiz, KH P906
Resanovic, I P1041
Resch, J OC3.2
Resende, E P235
Resmini, E OC4.4, P868 & P873
Rexhepi, A P253
Rey, R P645
Reyes-García, R P595
Reynolds, J P178
Rezaei, N P3
Rezza, A P659
Riaz, M P42, P44 & P852
Ribeiro, C P1101, P1136 & P40
Ribeiro, EB P746
Ricarte-Filho, J S16.3
Ricca, V P675
Ricci, B P648
Ricciato, MP P1049
Richelsen, B P638
Rideg, O P1000
Rietzschel, E P1015
Rindi, G MTE5, P532 & P561
Ring, T P875
Ringholm, L P416
Rios, E P243
Ripoll, RQ P377 & P564
Risbridger, G OC4.1
Rischel, C P627
Ristic, N P835 & P838
Risueño, EG P934
Rittig, S OC6.3
Ritzel, R S9.1
Rivera, NG P1112
Rivera, R P748
Rivero-Cortés, E P844
Rizk-Rabin, M P3 & P5
Rizos, D P187
Rizvi, S P44
Rizvi, SSR P337, P42 & P852
Roa, J P684
Roberto Salgado, L P841
Robertson, G S10.2
Robinson, S P156
Robles, AR P126
Roche, C P334 & S11.3
Rochira, V P1005, P1082, P1088, P1092, P640, P84 & P881
Roden, M P348
Rodríguez, IC P79 & P934
Rodríguez, JP P1112, P319, P444, P448, P479 & P491
Rodríguez-Gutiérrez, R P442
Rodrigo, ST P564
Rodrigues, E P1089, P128, P710 & P717
Rodrigues, F P1030, P163 & P472
Rodrigues, J P1084
Rodriguez Caballero, MG OC3.4
Rodriguez, A P1084 & P431
Rodriguez, I P777
Rodriguez, JE P1111
Rodriguez, M P1084
Rodriguez-Molina, JM P190
Rodriguez-Rodero, S OC3.4
Rodzina, E P433
Roed, H P877
Roef, G P1015 & P977
Roelofs, MJE P644
Roemmler, J P858 & P882
Rohani, Z P760
Rohner-Jeanrenaud, F P681
Roig-Espert, B P209 & P211
Rojas-García, P645
Rojo, G P133, P152 & P153
Rojo-Martínez, G P737
Roldán-Caballero, P P474, P56, P745 & P764
Roldan, P P493
Román, MM P606
Romano, D S11.3
Romanov, G P459
Romero, E P901
Romero, SG P1140
Roncati, L P1088 & P1092
Ronchi, C OC4.2 & OC4.3
Rondena, R P867
Rorato, R P736
Rosário, F P806
Rosa Pelizzo, M P1099
Rosca, R P104
Rosen, T P871
Rosenmai, AK S23.3
Rosenwald, A OC4.2
Ross, I P34
Ross, R EN2.2, P1, P2 & P218
Rossetti, R OC5.1
Rossi, M P653
Rossi, V P524
Rostomyan, L P527 & P846
Rotermund, R P909
Roupas, N P578
Roussel, HW P5
Roussel, R P637
Rousso, D P370
Rouxel, A P1013
Rovira, E P1030
Roxana, S P1074
Roy, S P359
Rozas-Moreno, P P595
Rozhinskaya, L P32, P527, P706, P832 & P845
Rozhko, A P433
Ruas, L P254, P421 & P954
Ruben, N P364
Rubin, B P1099, P17 & P20
Rubino, M P169
Rubio, E P133, P152 & P153
Rubio, M P774 & P775
Ruci, D P97
Rucz, K P1097
Rudas, L P320
Ruffilli, I P1081
Ruffin, M P845, P847 & P906
Ruige, J P182 & P770
Ruiz de Adana, M P152, P153, P312 & P768
Ruiz Gracia, T P708
Ruiz, I P714
Ruiz, M P275 & P793
Ruiz-Garcia, A P209 & P211
Ruiz-Llorente, L P722
Ruiz-Pino, F P684
Rump, L P8
Rungby, J P352, P358, P380 & P641
Runkle De La Vega, I P708
Rupa, R P839
Rusalenko, M P433
Ruscica, M P514 & P690
Russel, FGM P644
Russo, E OC3.5
Russo, G P687
Russo, MD P744
Rusu, C P903
Ruth, B P1054
Rutishauser, J P702
Rutten, E N1
Rutz, C P577
Rutz, S P842
Ruvolo, G P586
Ruyatkina, L P1057
Ruzsa, B P1000
Ryan Ardeña, GJ P197
Ryan, A OC4.1
Ryder, C N2
Ryder, R P899
Ryder, WDJ P965
Ryzhenkova, MI P1105

Šipr, K P171
Šiprová, H P171
Šumarac-Dumanović, M P761
Şen, H P263, P308 & P309
S Andersen, M P638
S Young, I P990
Sá, M P235
Sánchez, IP P1109 & P970
Sánchez-Tejada, L P844