



The contractility of circular and longitudinal intestinal smooth muscle is differentially altered during trinitrobenzenesulfonic acid induced ileitis in guinea pigs

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XV INTERNATIONAL SYMPOSIUM ON GASTROINTESTINAL MOTILITY

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THE CONTRACTILITY OF CIRCULAR AND LONGITUDINAL INTESTINAL SMOOTH MUSCLE IS DIFFERENTIALLY ALTERED DURING TRINITROBENZENESULFONIC ACID INDUCED ILEITIS IN GUINEA PIGS.
J.P. Martinolle, R. Garcia-Villar, J. Fioramonti, L. Buéno. INRA, Department of Pharmacology, BP.3, 31931, Toulouse, France.

Gut inflammation is accompanied by changes in intestinal contractility in both humans and animals. Intrajejunal instillation of trinitrobenzenesulfonic acid (TNBS) is an established animal model of chronic ileitis. This study was aimed at determining the effects of TNBS-induced inflammation on the motility of both circular and longitudinal smooth muscle layers of the guinea-pig ileum.

Methods: Guinea-pigs given a single intrajejunal administration (0.3 ml) of either saline (controls, n=6) or TNBS in 50% ethanol (80 mg/kg, n=6), were sacrificed 6 days later. Ileal strips (longitudinal and circular; 8 x 2 mm) were placed in a superfusion system (Krebs; 37°C; 2 ml/min). Concentration-response curves to carbachol, histamine and adrenaline were constructed (0.1 nM to 0.1 mM). Adrenaline was superfused in a solution containing alprenolol, with either yohimbine or prazosin (all 1 µM). Ileal isometric contractions were recorded and quantified. Maximal effects (Emax) for each agent were expressed in grams over baseline, per mm² of cross-sectional area (mean ± SEM; n=6). Drug potency was expressed as EC₅₀ in µM (mean ± SEM; n=6). Response to KCl (60mM) was taken as a standard control contraction.

Results: Longitudinal strips from controls were dose-dependently contracted by carbachol (Emax: 0.49±0.08), histamine (0.34±0.08), adrenaline+yohimbine (0.38±0.06) and adrenaline+prazosin (0.24±0.03). EC₅₀s were 0.23±0.19, 0.73±0.32, 17.2±8.3 and 23.5±12.6, respectively. In longitudinal strips from TNBS-treated animals, Emax for carbachol (0.84±0.11) and histamine (0.65±0.08) were significantly increased (p<0.05) without changes in EC₅₀s (0.11±0.07 and 0.98±0.37). In contrast there were no contractile responses to either α₁- or α₂-adrenoceptor stimulation by adrenaline solutions. Circular strips from controls were dose-dependently contracted by carbachol (1.09±0.15) and histamine (0.63±0.14) with EC₅₀s of 0.27±0.24 and 0.69±0.41, but never by adrenaline. In circular strips from TNBS-treated animals these Emax were not modified (0.95±0.14 and 0.52±0.09), but EC₅₀s were increased (7.20±2.30 and 7.40±1.87), while adrenaline remained without effect. Moreover, 60 mM KCl-induced contraction was not different in TNBS-treated tissues and in controls, neither in longitudinal nor in circular strips.

Conclusion: During TNBS-induced ileitis in guinea-pigs, the in vitro contractile responses of circular and longitudinal smooth muscle to receptor agonists is differentially altered, while nonreceptor-induced KCl contraction is not modified.

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HIGH FAT MEALS: DO THEY REALLY AFFECT THE COMPETENCE OF THE OESOPHAGO-GASTRIC JUNCTION?

M. Mangano, A. Picone, P.A. Bianchi, R. Penagini

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Although fatty foods are commonly considered detrimental in patients with gastro-oesophageal reflux (GOR) disease there are few and inconclusive data on the effect of fat on GOR and its mechanisms. It has been suggested that fat may provoke reflux by increasing the rate of transient lower oesophageal sphincter relaxations (TLOSRS) and their association with reflux and/or by decreasing basal lower oesophageal sphincter pressure (BLOSP). **Aim:** To compare the effect of a balanced meal (2500 kJ, 30% fat, 53% carbohydrate, 17% protein) and an equicaloric high fat meal (65% fat) on GOR, TLOSRS and BLOSP in 8 healthy subjects aged 19-30 years (6 men) and in 12 patients with GOR disease (erosive oesophagitis or oesophageal pH<4 for >5% of time) aged 23-57 years (9 men). All subjects and 7 patients were studied in the recumbent and 5 patients in the upright position. **Methods:** The 2 meals were administered on separate days in randomised order and combined oesophageal pH and motility monitoring (pharynx, oesophageal body, lower oesophageal sphincter with a sleeve, stomach) was performed for a period of 180 min after each meal.

Results (mean±SE):

Meal type	Healthy subjects		Patients *	
	Balanced	High fat	Balanced	High fat
% pH<4	2.7±0.9	2.6±1.9	15.4±4.6	12.9±5.0
Reflux episodes (no/h)	1.0±0.4	1.1±0.7	5.4±1.2	4.8±1.3
TLOSRS (no/h)	3.7±0.8	3.9±0.6	7.2±2.4	5.8±0.9
% TLOSRS with GOR	36±11	28±7	65±8	57±8
BLOSP (mmHg)	14±3	12±1	7±2	9±2

* Data from recumbent and upright patients were pooled because posture had no influence on results.

Conclusions: Doubling the fat content of a meal does not affect postprandial oesophago-gastric competence or GOR for at least 3 hours in either healthy subjects or patients with GOR disease. The possibility remains that fat increases GOR in the late post-prandial period by prolonging the time acidic material persists in the stomach.

THE CONTRACTILITY OF CIRCULAR AND LONGITUDINAL INTESTINAL SMOOTH MUSCLE IS DIFFERENTIALLY ALTERED DURING TRINITROBENZENESULFONIC ACID INDUCED ILEITIS IN GUINEA PIGS.

J.P. Martinolle, R. Garcia-Villar, J. Fioramonti, L. Bueno. INRA, Department of Pharmacology, BP.3, 31931, Toulouse, France.

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Results: Longitudinal strips from controls were dose-dependently contracted by carbachol (Emax: 0.49±0.08), histamine (0.34±0.08), adrenaline+yohimbine (0.38±0.06) and adrenaline+prazosin (0.24±0.03). EC50s were 0.23±0.19, 0.73±0.32, 17.2±8.3 and 23.5±12.6, respectively. In longitudinal strips from TNBS-treated animals, Emax for carbachol (0.84±0.11) and histamine (0.65±0.08) were significantly increased (p<0.05) without changes in EC50s (0.11±0.07 and 0.98±0.37). In contrast there were no contractile responses to either α1- or α2-adrenoceptor stimulation by adrenaline solutions. Circular strips from controls were dose-dependently contracted by carbachol (1.09±0.15) and histamine (0.63±0.14) with EC50s of 0.27±0.24 and 0.69±0.41, but never by adrenaline. In circular strips from TNBS-treated animals these Emax were not modified (0.95±0.14 and 0.52±0.09), but EC50s were increased (7.20±2.30 and 7.40±1.87), while adrenaline remained without effect. Moreover, 60 mM KCl-induced contraction was not different in TNBS-treated tissues and in controls, neither in longitudinal nor in circular strips.

Conclusion: During TNBS-induced ileitis in guinea-pigs, the in vitro contractile responses of circular and longitudinal smooth muscle to receptor agonists is differentially altered, while nonreceptor-induced KCl contraction is not modified.

MECHANISMS OF INCREASED GASTRIC EMPTYING BY ERYTHROMYCIN

C. Mathis and C.H. Malbert INRA, Station de Recherches Porcines, 3559 Saint Gilles, L'Hermitage, France

Gastric emptying rate depends on the ratio between propulsive and resistive forces, the later being mainly generated by the pylorus. Yet, increased resistance is supposed to retard gastric emptying, some gastrokinetic compounds such as cisapride also increase pyloric resistance. We have investigated changes in transpyloric flow and pyloric resistance induced by erythromycin (lactobionate, EM) in eight chloralose-urethane anaesthetised pigs. EM (10⁻³M) or saline was infused during 30 min in the gastropiploic artery cannulated in caudad direction 10 cm before the pylorus. Antropyloroduodenal motility was measured with a sleeve/side holes assembly and transpyloric flow was evaluated with a juxtapyloric flowmeter probe. Increased gastric emptying after EM was the consequence of flow pulses of larger stroke volume (13.7 ± 2.24 vs. 3.6 ± 0.30 ml, p<0.05) and of longer duration (10.9 ± 0.45 vs. 6.9 ± 0.23 s, p<0.05) but occurring at the same frequency (3.9 ± 0.55 vs. 3.2 ± 0.30 per min, p>0.05). Duodeno-gastric backflow that amounted to 9% of forward flow with saline, was absent with EM. Amplitude and frequency of the antropyloroduodenal pressure events were doubled irrespective of the recording site (57.2 ± 4.78 vs. 38.2 ± 2.52 mmHg and 4.3 ± 0.17 vs. 3.0 ± 0.11 per min for the pylorus, p<0.05). They were associated to flow pulses in a 1:1 ratio instead of a 1:2 ratio with saline. EM induced a significant decrease in pyloric resistance that was halved compared to saline (2.3 ± 0.21 vs. 4.6 ± 0.46 mmHg.ml⁻¹.s, p<0.05), a consequence of a reduced time interval between the onset of the pyloric pressure events and the onset of the flow pulses (1.0 ± 0.31 vs. 3.2 ± 0.58 s, p<0.05) rather than decrease in pyloric basal tone that was actually increased by EM (34.1 ± 2.07 vs. 18.2 ± 0.69 mmHg for EM vs. saline). In conclusion, EM increases gastric emptying rate by generating flow pulses of greater stroke volume and of unchanged frequency. The greater stroke volume of the flow pulses originates from a reduced pyloric resistance.

INCREASED GASTRIC EMPTYING RATE BY ERYTHROMYCIN IS VAGALLY MEDIATED

C. Mathis and C.H. Malbert INRA, Station de Recherches Porcines 35590 Saint Gilles, L'Hermitage, France

Motilin receptor density and affinity suggest that erythromycin (EM) stimulates gastric motility and emptying by a combined action on the smooth muscle and myenteric neurones. However, because Motilin receptors have recently been found in the brain and on vagal related structures, the central nervous system might also be involved in EM *in vivo* activity. The possible modulation of EM effects by the vagus has been investigated in eight chloralose-urethane anaesthetised pigs. EM (lactobionate, 10⁻³M) was infused for 30 min in the gastropiploic artery that was cannulated in caudad direction 10 cm before the pylorus. Reversible interruptions of vagal pathways were performed by repetitive cooling of the cervical vagal trunks at 3°C during EM infusion. Antropyloroduodenal motility was evaluated with a sleeve/side holes assembly and transpyloric flow was measured by a juxtapyloric flowmeter probe. Vagal cooling produced a marked inhibition in EM induced increased gastric emptying (23 ± 1.1 vs. 50 ± 2.6 ml.min⁻¹, p<0.05). Gastric emptying remained pulsatile and the frequency of the flow pulses was unchanged, but their stroke volume was halved (7.8 ± 3.31 vs. 13.7 ± 2.24 ml, p<0.05). This was the consequence of flow pulses of reduced duration (8.1 ± 0.77 vs. 10.9 ± 0.45 s, p<0.05) and of smaller peak flow rate (2.8 ± 0.71 vs. 4.4 ± 0.53 ml.s⁻¹, p<0.05). On the contrary, vagal cooling did not significantly modify the increased frequency and amplitude of the antropyloroduodenal pressure events induced by EM. The decrease emptying rate during vagal cooling was due to a threefold increase in pyloric resistance (6.3 ± 1.98 vs. 2.3 ± 0.21 mmHg. ml⁻¹.s, p<0.05), a consequence of a longer time interval between the onset of pyloric pressure events and transpyloric flow pulses (1.0 ± 0.31 vs. 1.9 ± 0.33 s). It is concluded that (i) EM related increased emptying rate is dependant on the integrity of vagal pathways and (ii) vagally mediated EM effects are directed towards pyloric resistance and not gastric propulsive forces.

XV International Symposium on Gastrointestinal Motility

AUTHOR INDEX

- van Aalst K, 271
 Abrahamson H, 250
 Adams RJ, 280
 Adam MB, 285
 Adreassen D, 290
 Alkermans LMA, 267
 Allcher HD, 261, 268
 Andrioli A, 252
 Angel F, 246, 248
 Angel I, 249, 276
 Anton S, 268
 Arcidiacono M, 254
 Arhan P, 268
 Arruho P, 261
 Babyn PS, 250
 Bader R, 256
 Bachli D, 246
 Bakewell M, 277
 Balloni M, 247
 Barbars C, 253, 254
 Barbars M, 290
 Barbars JP, 246
 Bartelli E, 246
 Barlow J, 255
 Basilio G, 276
 Bass P, 287
 Bassotti G, 254
 Bauer AJ, 246, 251
 Bauleit E, 280
 Bayguinov O, 250
 Bayguinov YR, 262
 Bazzocchi G, 247
 Bednarz S, 279
 Berzin I, 251, 270
 Bharucha AE, 249
 Bianchi PA, 273
 Bionason E, 250
 Blackshaw LA, 247, 269
 Blackshaw PE, 263
 Blackshaw CE, 254, 255
 Boeckxstaen G, 248
 Boninelli G, 248
 Bonino R, 278
 Bonneville JL, 274
 Bordet JR, 246, 248
 Borcia A, 282
 Boulant J, 248
 Boukurt T, 278
 Brashers HR, 250
 Brice MR, 248, 275
 Brown BP, 248, 286
 de Bruin H, 251
 Buchheit KH, 280
 Bufo L, 249, 257, 259, 264, 273, 280, 282
 Buhl T, 280
 Burns AKI, 249, 262, 287
 Bywater RAR, 258, 288
 Camilleri M, 249
 Candura SM, 253
 Cassa SD, 249, 250
 Carini M, 257
 Carli A, 250
 de Carle DJ, 252
 Carlos MA, 250
 Cardiel M, 250
 Castillo FD, 277
 Cayabyab FS, 251
 Chavarrat R, 247
 Chen JDZ, 251, 269
 Chey WD, 262, 266
 Chivon M, 246
 Chiarelli C, 275
 Chignell K, 287
 Chittum MO, 259, 272
 Chrysos E, 290
 Cicalea M, 278
 Cicalea L, 251
 Cicciocioppo AF, 257, 260
 Clayton NM, 252
 Clemens A, 265
 Cleveland S, 284
 Collman PJ, 252
 Congard P, 268
 Contesani Avesani E, 276
 Conti E, 247
 Cook IL, 252
 Cook MA, 276
 Cooke HJ, 293
 Corazzini E, 246, 261, 278
 Correas G, 252, 287
 Conalderi R, 253, 254
 Cortesini C, 257
 Costa E, 278
 Coult B, 253, 289, 291
 Coyle J, 287
 Craig JW, 274
 Crema A, 254
 Cucci T, 254
 Cuche G, 253
 Custer-Hagen T, 286
 D'Altoche L, 280
 D'Amato M, 248, 258
 Daniel EE, 251
 Dapporto G, 277
 Davidson C, 257
 De Ciau P, 255, 283
 De Giorgio R, 253, 254
 De Looze D, 254
 De Man JG, 254, 255
 De Mynck W, 254
 De Ponti F, 254
 De Winter BY, 254, 255
 Dedrichs E, 278
 Delvaux M, 264, 282
 Denel G, 287
 Dent J, 247, 269, 277
 Depoortere I, 255, 291
 Camilleri M, 249
 Candura SM, 253
 Cassa SD, 249, 250
 Carini M, 257
 Carli A, 250
 de Carle DJ, 252
 Carlos MA, 250
 Cardiel M, 250
 Castillo FD, 277
 Cayabyab FS, 251
 Chavarrat R, 247
 Chen JDZ, 251, 269
 Chey WD, 262, 266
 Chivon M, 246
 Chiarelli C, 275
 Chignell K, 287
 Chittum MO, 259, 272
 Chrysos E, 290
 Cicalea M, 278
 Cicalea L, 251
 Cicciocioppo AF, 257, 260
 Clayton NM, 252
 Clemens A, 265
 Cleveland S, 284
 Collman PJ, 252
 Congard P, 268
 Contesani Avesani E, 276
 Conti E, 247
 Cook IL, 252
 Cook MA, 276
 Cooke HJ, 293
 Corazzini E, 246, 261, 278
 Correas G, 252, 287
 Conalderi R, 253, 254
 Cortesini C, 257
 Costa E, 278
 Coult B, 253, 289, 291
 Coyle J, 287
 Craig JW, 274
 Crema A, 254
 Cucci T, 254
 Cuche G, 253
 Custer-Hagen T, 286
 D'Altoche L, 280
 D'Amato M, 248, 258
 Daniel EE, 251
 Dapporto G, 277
 Davidson C, 257
 De Ciau P, 255, 283
 De Giorgio R, 253, 254
 De Looze D, 254
 De Man JG, 254, 255
 De Mynck W, 254
 De Ponti F, 254
 De Winter BY, 254, 255
 Dedrichs E, 278
 Delvaux M, 264, 282
 Denel G, 287
 Dent J, 247, 269, 277
 Depoortere I, 255, 291
 Kadowaki M, 265
 Kato H, 285
 Kahlil PJ, 269, 281
 Kang YW, 265
 Karas M, 265
 Katschinski M, 285
 Katsoulis S, 265
 Keef KD, 266
 Kennedy MD, 252
 Kiernan HD, 266
 Kim KS, 267
 Kim M, 262, 266
 Kirchgessner A, 266
 Klatt M, 267
 Klein A, 246
 Kletter K, 261
 Kobayashi S, 287
 Koh SD, 267
 Konoh A, 265
 Konurek JW, 279
 Krause J, 267
 Kroeckel ABA, 267
 Kuhn K, 280
 Kuramoto H, 267
 Kurjak M, 268
 Messoni E, 253
 Metman EH, 246, 280
 Mettemans AL, 248
 Miampamba M, 275
 Midvedt T, 264
 Miki K, 277
 Mikkelsen HR, 272, 275, 290
 Miller AJ, 279
 Miller SM, 271, 277
 Minasi JS, 285
 Minni E, 254
 Miral RA, 270, 275
 Mizumoto A, 286
 Moneta NA, 276
 Moore AA, 250
 Morand R, 276
 Morelli A, 254
 Morris J, 259
 Morris SC, 260
 Morry-Wortmann C, 265
 Muelemans AL, 275
 Mueller-Stoeber S, 278
 Muir TC, 274, 281
 Mularczyk A, 276
 Muth F, 276
 Mutillo D, 261
 Murray DC, 266
 Naccarato R, 260
 Nagakura Y, 265
 Neild PJ, 277
 Neumann R, 265
 Nguyen HN, 277
 Nitceki S, 277
 O'Brien R, 287
 O'Connor MK, 281
 Oliver M, 290
 Oliver MR, 275
 Onari T, 247, 277
 Orth KH, 278
 Oster-Jorgensen E, 281
 Ouyang C, 262, 266, 289
 Ghoo Y, 253, 271
 Gellert C, 254
 Gielch PJ, 263
 Giall M, 260
 Gille B, 285
 Goldhill JM, 260, 263
 Goldworthy W, 277
 Goncalves E, 248, 258, 283
 Gregersen H, 292
 Grossi L, 257, 260
 Grundy D, 256
 Guftin S, 253, 269
 Guerrero-Lindner E, 261
 Habib EL, 246, 261
 Haga N, 286, 293
 Hagel S, 263
 Hahn A, 261
 Hannum J, 265
 Hannum J, 261
 Hanzel KE, 265
 Hansen M, 262, 271
 Hanson RE, 249
 Hanson RW, 262
 Hanzel KW, 262
 Harty GJ, 277
 Haslam R, 277
 Hasler W, 262, 266
 Hatfield R, 282
 Hauechmidt B, 285
 Haupt JA, 247, 269
 Haussinger D, 259
 Hayes J, 263
 Hayden JM, 263
 Helleron P, 264, 271, 274
 Helber LF, 275
 Hendel L, 292
 Herman AG, 254, 255
 Hermann R, 248, 286
 Hillsley K, 256
 Leroy F, 268
 Hogan WJ, 268, 274
 Hollstein K, 267
 Holst JJ, 282
 Horowitz B, 292
 Horvitz J, 263
 Horvitz M, 285
 Houghton LA, 259
 Hoyer D, 280
 Huber A, 268
 Huzinga J, 290
 Huzinga JD, 263, 270, 272
 Husebye E, 264, 274
 Hwang JS, 265
 Itoh Z, 286, 293
 Jancos J, 292
 Janssens J, 252, 253, 274, 287, 289, 291
 Jean A, 252
 Jernstam O, 264
 Jimenez M, 248, 251, 283
 Joliba E, 288
 Johnson AG, 272
 Johnson CF, 284, 286
 Jondrick R, 264
 Juncas C, 283
 Julia V, 264
 Kadowaki M, 265
 Kato H, 285
 Kahlil PJ, 269, 281
 Kang YW, 265
 Karas M, 265
 Katschinski M, 285
 Katsoulis S, 265
 Keef KD, 266
 Kennedy MD, 252
 Kiernan HD, 266
 Kim KS, 267
 Kim M, 262, 266
 Kirchgessner A, 266
 Klatt M, 267
 Klein A, 246
 Kletter K, 261
 Kobayashi S, 287
 Koh SD, 267
 Konoh A, 265
 Konurek JW, 279
 Krause J, 267
 Kroeckel ABA, 267
 Kuhn K, 280
 Kuramoto H, 267
 Kurjak M, 268
 Messoni E, 253
 Metman EH, 246, 280
 Mettemans AL, 248
 Miampamba M, 275
 Midvedt T, 264
 Miki K, 277
 Mikkelsen HR, 272, 275, 290
 Miller AJ, 279
 Miller SM, 271, 277
 Minasi JS, 285
 Minni E, 254
 Miral RA, 270, 275
 Mizumoto A, 286
 Moneta NA, 276
 Moore AA, 250
 Morand R, 276
 Morelli A, 254
 Morris J, 259
 Morris SC, 260
 Morry-Wortmann C, 265
 Muelemans AL, 275
 Mueller-Stoeber S, 278
 Muir TC, 274, 281
 Mularczyk A, 276
 Muth F, 276
 Mutillo D, 261
 Murray DC, 266
 Naccarato R, 260
 Nagakura Y, 265
 Neild PJ, 277
 Neumann R, 265
 Nguyen HN, 277
 Nitceki S, 277
 O'Brien R, 287
 O'Connor MK, 281
 Oliver M, 290
 Oliver MR, 275
 Onari T, 247, 277
 Orth KH, 278
 Oster-Jorgensen E, 281
 Ouyang C, 262, 266, 289
 Janssens J, 252, 253, 274, 287, 289, 291
 Jean A, 252
 Jernstam O, 264
 Jimenez M, 248, 251, 283
 Joliba E, 288
 Johnson AG, 272
 Johnson CF, 284, 286
 Jondrick R, 264
 Juncas C, 283
 Julia V, 264
 Kadowaki M, 265
 Kato H, 285
 Kahlil PJ, 269, 281
 Kang YW, 265
 Karas M, 265
 Katschinski M, 285
 Katsoulis S, 265
 Keef KD, 266
 Kennedy MD, 252
 Kiernan HD, 266
 Kim KS, 267
 Kim M, 262, 266
 Kirchgessner A, 266
 Klatt M, 267
 Klein A, 246
 Kletter K, 261
 Kobayashi S, 287
 Koh SD, 267
 Konoh A, 265
 Konurek JW, 279
 Krause J, 267
 Kroeckel ABA, 267
 Kuhn K, 280
 Kuramoto H, 267
 Kurjak M, 268
 Messoni E, 253
 Metman EH, 246, 280
 Mettemans AL, 248
 Miampamba M, 275
 Midvedt T, 264
 Miki K, 277
 Mikkelsen HR, 272, 275, 290
 Miller AJ, 279
 Miller SM, 271, 277
 Minasi JS, 285
 Minni E, 254
 Miral RA, 270, 275
 Mizumoto A, 286
 Moneta NA, 276
 Moore AA, 250
 Morand R, 276
 Morelli A, 254
 Morris J, 259
 Morris SC, 260
 Morry-Wortmann C, 265
 Muelemans AL, 275
 Mueller-Stoeber S, 278
 Muir TC, 274, 281
 Mularczyk A, 276
 Muth F, 276
 Mutillo D, 261
 Murray DC, 266
 Naccarato R, 260
 Nagakura Y, 265
 Neild PJ, 277
 Neumann R, 265
 Nguyen HN, 277
 Nitceki S, 277
 O'Brien R, 287
 O'Connor MK, 281
 Oliver M, 290
 Oliver MR, 275
 Onari T, 247, 277
 Orth KH, 278
 Oster-Jorgensen E, 281
 Ouyang C, 262, 266, 289
 Janssens J, 252, 253, 274, 287, 289, 291
 Jean A, 252
 Jernstam O, 264
 Jimenez M, 248, 251, 283
 Joliba E, 288
 Johnson AG, 272
 Johnson CF, 284, 286
 Jondrick R, 264
 Juncas C, 283
 Julia V, 264
 Kadowaki M, 265
 Kato H, 285
 Kahlil PJ, 269, 281
 Kang YW, 265
 Karas M, 265
 Katschinski M, 285
 Katsoulis S, 265
 Keef KD, 266
 Kennedy MD, 252
 Kiernan HD, 266
 Kim KS, 267
 Kim M, 262, 266
 Kirchgessner A, 266
 Klatt M, 267
 Klein A, 246
 Kletter K, 261
 Kobayashi S, 287
 Koh SD, 267
 Konoh A, 265
 Konurek JW, 279
 Krause J, 267
 Kroeckel ABA, 267
 Kuhn K, 280
 Kuramoto H, 267
 Kurjak M, 268
 Messoni E, 253
 Metman EH, 246, 280
 Mettemans AL, 248
 Miampamba M, 275
 Midvedt T, 264
 Miki K, 277
 Mikkelsen HR, 272, 275, 290
 Miller AJ, 279
 Miller SM, 271, 277
 Minasi JS, 285
 Minni E, 254
 Miral RA, 270, 275
 Mizumoto A, 286
 Moneta NA, 276
 Moore AA, 250
 Morand R, 276
 Morelli A, 254
 Morris J, 259
 Morris SC, 260
 Morry-Wortmann C, 265
 Muelemans AL, 275
 Mueller-Stoeber S, 278
 Muir TC, 274, 281
 Mularczyk A, 276
 Muth F, 276
 Mutillo D, 261
 Murray DC, 266
 Naccarato R, 260
 Nagakura Y, 265
 Neild PJ, 277
 Neumann R, 265
 Nguyen HN, 277
 Nitceki S, 277
 O'Brien R, 287
 O'Connor MK, 281
 Oliver M, 290
 Oliver MR, 275
 Onari T, 247, 277
 Orth KH, 278
 Oster-Jorgensen E, 281
 Ouyang C, 262, 266, 289