Eukaryotic Lipids;

Treasure of Regulatory

Information

FEBS/IUBMB Workshop

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Poster abstracts

Poster abstracts (alphabetic)

The numbering refer to the numbers of the poster boards.
The presenter's name is underlined and only her/his lab is mentioned.
[#] *Selected for oral presentation.*

1. **Amara,** Sawsan
2. **Angelini,** Roberto
3. **Ayvazyan,** Naira
4. **Baksa,** Attila
5. [#]**Balreira,** Andrea
6. **Baranowski,** Marcin
7. **Barriga,** Hanna
8. **Belter,** Agnieszka
9. **Bilgin,** Mesut
10. [#]**Bohdanowicz,** Michal
11. [#]**Borroni,** Maria
12. **Bozkurt,** Ozlem
13. **Brioschi,** Loredana
14. **Cano,** Ainara
15. **Cerrada de Dueñas,** Alejandro
16. **Chiritoiu,** Gabriela
17. **Delage,** Elise
18. **Ducheix,** Simon
19. **Gale,** Elikana
20. **Garner,** Kathryn
21. **Gsell,** Martina
22. **Gutiérrez Martinez,** Enric
23. **Hermann,** Sandra
24. **Jaafar,** Rami
25. [#]**Jaikishan,** Shishir
26. [#]**Kang,** Hye Won
27. **Kania,** Madalena
28. [#]**Kasza,** Ildikó
29. **Kjellberg,** Matti
30. **Kosicek,** Marko
31. ***Kreim,** Martin
32. **Lissina,** Elena
33. **Macedo,** Fatima
34. **Manni,** Marco
35. **Marin,** Mari
36. **Martin,** Mauricio
37. **Maula,** Terhi
38. **Mazid,** Abdul
39. **Mohamed,** Amany
40. **Montagner,** Alexandra
41. **Mørch Frøsig,** Merethe
42. **Olssen,** Petter
43. **Oresti,** Gerardo M.
44. **Ota,** Katja
45. **Poeschl,** Margret
46. [#]**Ruggles,** Kelly
47. [#]**Serricchio,** Mauro
48. **Telenius,** Jelena
49. **Tosheska-Trajkovska,** Katerina
50. **Weis,** Nicole
51. **Wilmes,** Claudia
52. **Wu,** Wen-Guey
53. **Zhao,** Hongxia

No poster

Heilmeyer, prof.dr. L., emeritus

11. #Cholesterol levels determine AChR endocytic route in CHO-K1/A5 cells

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Stability of the nicotinic acetylcholine receptor (AChR) at the cell surface is critical to the correct functioning of the cholinergic synapse. Cholesterol (Chol) is an essential lipid that modulates AChR levels at the plasmalemma and ion translocation. We have studied the endocytosis of AChR in CHO-K1/A5 cells, a Chinese hamster ovary (CHO) cell line heterologously expressing murine muscle adult-type receptor under different Chol membrane content. Contrary to the norm, endocytosis of cell-surface AChR is accelerated by membrane Chol depletion via a hitherto unknown mechanism. This acceleration is no longer operative when membrane Chol levels are restored. We explored the possible mechanism involved in receptor loss in Chol-depleted cells (Chol-). Under such conditions the AChR is internalized by a ligand-, clathrin- and dynamin-independent mechanism, which does not involve the presence of the AChR-associated protein rapsyn. The small GTPase Rac1 is required: expression of a dominant negative form of Rac1, Rac1N17, abrogates receptor endocytosis. At variance with the endocytic pathway in control CHO cells, the accelerated AChR internalization proceeds even upon disruption of the actin cytoskeleton and does not depend on the cytoskeleton-associated inositol lipid PI(4,5)P2; its sequestration by the PH domain of phospholipase C does not alter endocytosis. AChR internalization under Chol- conditions is furthermore found to require the activity of Arf6 and its effectors Rac1 and phospholipase D. Thus, membrane Chol appears to act as a key homeostatic regulator of cell-surface receptor levels, determining the rate and mechanism of AChR endocytosis.