

NRC · CNRC

Canadian Journal of
Physiology and Pharmacology

Revue canadienne de
physiologie et pharmacologie

Abstracts

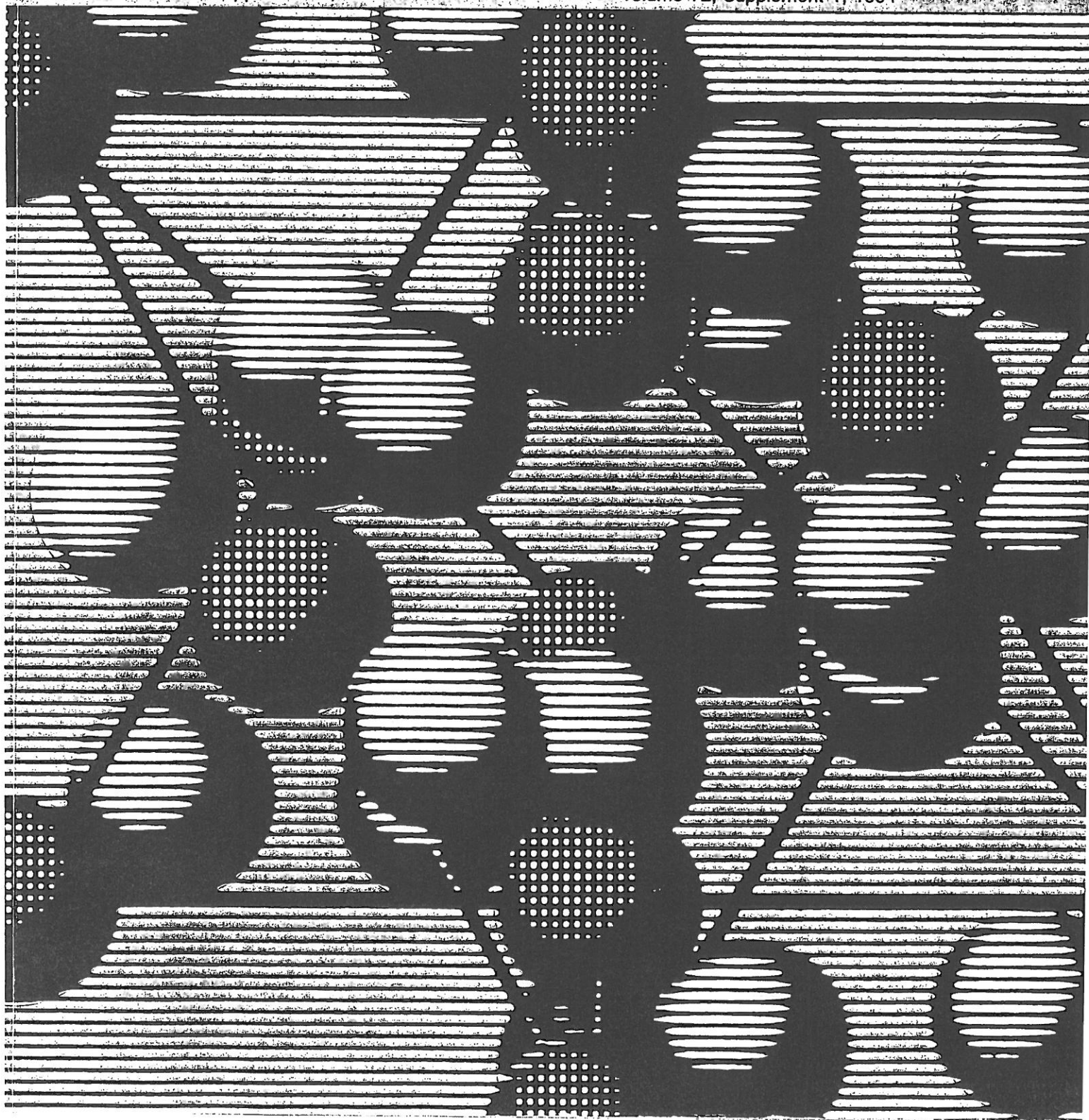
**XIIth International
Congress of Pharmacology
24 – 29 July 1994, Montréal, Canada**

Volume 72, Supplement 1, 1994

Résumés

**XII^e Congrès
international de pharmacologie
du 24 au 29 juillet 1994, Montréal, Canada**

Volume 72, Supplément 1, 1994





XIIth International Congress of Pharmacology XII^e Congrès international de pharmacologie

July / juillet 24 - 29, 1994, Montréal, Québec, Canada

April 5, 1994

Chairman / Président
G.S. Marks
Queen's University

**Vice-Chairman /
Vice-président**
R. Capek
McGill University

Treasurer / Trésorier
A.W. Ford-Hutchinson
Merck Frosst Canada Inc.

Secretary / Secrétaire
P. du Souich
Université de Montréal

**Scientific Program /
Programme scientifique**
B. Collier
McGill University

**Commercial Exhibitor /
Exposition commerciale**
S. Katz
University of British
Columbia

**Local Organizing
Committee Chairman
Président du comité local**
M.G. Côté
Université de Montréal

**Satellite Symposia
Réunions satellites**
K.W. Renton
University of
Glasgow

Publication
A.C. Cuervo
McGill University

**Congress Director /
Directeur du congrès**
L. Forget
National Research
Council Canada /
Conseil national de
recherches Canada

**Information /
Renseignements**
Nicole A. Sarault
Congress Manager
Gestionnaire du congrès
National Research
Council Canada /
Conseil national de
recherches Canada
Ottawa, ON, Canada
K1A 0R6
Tel: (613) 993-7271
Fax: (613) 957-9828
Telex: 053-3145

MITSUKO SHINOHARA
DEPARTMENT OF PHARMACOLOGY
OSAKA DENTAL UNIVERSITY
5-31, OTEMAE 1-CHOME
CHUO-KU, OSAKA 540,
JAPAN

Dear Colleague:

We are pleased to inform you that your abstract has been accepted for a poster presentation at the XIIth International Congress of Pharmacology to be held on July 24-29, 1994 in Montréal, Québec.

Your abstract has been assigned to the poster session as follows:

Title: EFFECT OF BIO-NORMALIZER ON MACROPHAGE CHEMO-
TAXIS IN NATURALLY OCCURRING GINGIVITIS RAT, M.
Shinohara¹, Y. Morikawa¹, K. Ogata¹, A.J. Osato², K. Ohura¹,
¹Osaka Dental University, Osaka and ²Osato Research Institute, Gifu,
Japan

Abstract No: P10.5.003

Date of presentation: WEDNESDAY, July 27, 1994

Your poster board number will appear in the Final Program which you will receive at the registration desk.

The poster sessions will take place in the Exhibit Hall of the Palais des Congrès between 12:00 and 14:00 hours and the provided poster boards are approximately 1 m high and 1.5 m wide (please see preliminary program for details).

P10.5.1

GENERATION OF LEUKOTRIENE C₄ FAMINE RELEASE WITH PHOSPHATIDIC ACID VIA CALCIUM INFLUX IN MASTOCYTOMA CELL LINE.
Y. Moriya and H. Teshigawara. Department of pharmacology, Nihon University School of Dentistry at Matsudo, Matsudo, Chiba, Japan.

Mechanisms of phosphatidic acid (PA)-induced histamine release from mastocytoma P-815 cells were studied. Extracellular PA (1-100 μ M) induced histamine release and LTC₄ accumulation inside cells in dose dependent manner, but other phospholipids were not released histamine or released only slightly. LTC₄ accumulation was not significantly increased with addition of other phospholipids. When intracellular leukotriene C₄ (LTC₄) increased with addition of extracellular LTC₄, the release of histamine was observed. LTC₄-induced histamine release was inhibited with removal of extracellular Ca²⁺. Treatment with 5 μ M p-bromophenacyl bromide as a PLA₂ inhibitor significantly suppressed the increase of intracellular LTC₄ and PA-induced histamine release in dose dependent manner, even in the presence of PA. PA-induced histamine release and LTC₄ accumulation were inhibited with inorganic and organic Ca²⁺ channel blockers. Removal of extracellular Ca²⁺ also suppressed both LTC₄ accumulation and histamine release. When cells were incubated with high K⁺ solution (50mM), LTC₄ accumulation and histamine release were induced.

Our results indicated that intracellular LTC₄ was increased with PA via PLA₂ activation induced by Ca²⁺ influx and histamine release was elicited. Furthermore, Ca²⁺ channels on mastocytoma cells were regulated with PA. Ca²⁺ influx was required for the increase of intracellular LTC₄ and LTC₄-induced histamine release, respectively. There may be at least two pathways for PA-induced histamine release; it is known that PA provokes histamine release via diacylglycerol as a PA metabolite and a second messenger, another pathway is that PA elicits histamine release via the increase of intracellular LTC₄.

P10.5.3

EFFECT OF BIO-NORMALIZER ON MACROPHAGE CHEMOTAXIS IN NATURALLY OCCURRING GINGIVITIS RAT.
M. Shinohara, Y. Morikawa, K. Ogata, A.J. Osato* and K. Ohura.
Department of Pharmacology, Osaka Dental University, Osaka 540, *Osato Research Institute, Gifu 500, Japan.

Bio-catalyzer (Bio-normalizer: BN, Sun-O International Inc., Japan) is a white, sweet, multiple plant enzyme granules produced by fermentation of Carica papaya and tropical herbal plants with fermentative cereals from tropical and Japanese traditional food. BN is a free radical scavenger, which inhibits bacterial growth, and the action of toxohormone-L. In this study, we examined its effect on chemotaxis of macrophages (M Φ s) in naturally occurring gingivitis rat (ODUS/Odu). ODUS/Odu were given 2% BN for 2 months. Rat M Φ s were collected 4 days after intraperitoneal injection of 1% oyster glycogen solution in saline. M Φ chemotaxis was measured by the membrane filter method using 48-well microchemotaxis chambers, using zymosan-activated serum as the chemo-attractant. M Φ chemotaxis was significantly lower in the BN-treated group than in the control group. This suggests that BN plays an immunological role in host defence.

P10.5.5

EFFECT OF LPS ON BODY TEMPERATURE AND HYPOTHALAMIC PGE₂ PRODUCTION IN YOUNG AND OLD RATS
J. Kaplanski, V. Fraifeld and L. Abramovich. Department of Clinical Pharmacology, Ben-Gurion University of the Negev, Beer-Sheva, Israel.

Pyrogen-induced elevation of hypothalamic (HT) PGE₂ production is considered to play a major role in changing the thermoregulatory "set point" to a new level, that is the characteristic feature of fever. Aging is associated with attenuated febrile response. It was the aim of the present study to compare the effects of bacterial endotoxin (E. coli LPS, 50 μ g per animal, i. p.) on rectal temperature (RT) and ex-vivo HT PGE₂ production in young adult (4-6 mo-old) and old (24 mo-old) female Wistar rats. Animals were kept at 22 \pm 1 $^{\circ}$ C, 12L:12D, water and food ad libitum. RT was measured before and at different post-injection time periods. 24 h later, hypothalamus was excised and incubated in Krebs buffer solution for 3 h. The incubation medium was replaced each hour for PGE₂ assay by RIA. As compared with young adults, LPS-treated old rats displayed delayed febrile response, however no age-related differences in maximum RT amplitude were found (+RT, 6 h, 0.8 and 0, p<0.05; 24 h, 0.7 and 0.6 $^{\circ}$ C, young adults and olds, respectively). Both in young adult and old rats, LPS induced about two-fold elevation in HT PGE₂ production, as compared with control rats (p<0.05). Regardless of the lack of age-related differences in RT and HT PGE₂ production, measured 24 h post LPS injection, in young adult rats, in contrast with olds, correlation between RT changes and HT PGE₂ production was observed (p<0.05). The data obtained point toward probable disregulation between central and peripheral thermoregulatory mechanisms of fever in aging.

P10.5.2

EFFECT OF LPS AND INDOMETHACIN ON SHORT TERM FEVER RESPONSE AND HYPOTHALAMIC PGE₂ PRODUCTION IN MICE
L. Abramovich, V. Fraifeld, D. Shemi and J. Kaplanski. Department of Clinical Pharmacology, Ben Gurion University of the Negev, Beer Sheva, Israel.

Bacterial endotoxin (lipopolysaccharide, LPS) is a pyrogen that causes fever in a variety of animals. In our previous study we have demonstrated that in mice rectal temperature (RT) significantly decreased (p<0.01) during 6 hr after LPS injection, then followed recovery of RT to the control level up to 24 hr. In spite of the lack of hyperthermic phase, ex-vivo HT PGE₂ production, measured 24 hr after LPS injection, was two-fold greater as compared with controls. The present investigation was undertaken to correlate the effect of LPS and INDO on RT and HT PGE₂ production in the hypothalamic phase. Adult male mice in control room (22 \pm 1 $^{\circ}$ C) were injected with LPS (E. coli, 50 μ g/mice), saline and INDO (50 mg/kg) immediately after LPS. Significant decrease (p<0.01) in RT was found after LPS injection (3 hr, -1.5 $^{\circ}$ C), without changes in HT PGE₂ production. Effect of INDO+LPS on RT did not differ from that of LPS. However, in contrast to LPS, INDO significantly (p<0.01) decreased HT PGE₂ production in LPS-treated mice (control 91 \pm 3.8, LPS 94 \pm 6.0, INDO+LPS 34 \pm 2.7 pg/mg tissue/hr). Our data indicate, that no correlation occurs between HT PGE₂ production and changes in RT during initial hypothalamic phase. In conclusion, it seems that changes in RT of LPS-treated mice in the initial phase are not PGE₂-dependent.

P10.5.4

EFFECT OF STREPTOZOTOCIN-INDUCED DIABETES ON LEUKOCYTE FUNCTIONS IN RATS WITH NATURALLY OCCURRING GINGIVITIS.
K. Ohura, K. Ogata and M. Shinohara. Department of Pharmacology, Osaka Dental University, Osaka 540 Japan.

Many studies have shown that periodontal lesions in diabetic patients progress rapidly and resist treatment. To elucidate the effect of diabetes on periodontal lesions, chemotaxis, phagocytosis and superoxide anion (O₂⁻) production of leukocytes were studied in rats with naturally occurring gingivitis (ODUS/Odu) treated with streptozotocin to induce diabetes. Rat macrophages (M Φ s) were obtained 72 hours after intraperitoneal injection of mineral oil. M Φ chemotaxis was measured by the membrane filter method. Phagocytosis was measured using opsonized zymosan and fetal bovine serum. O₂⁻ production was measured by the reduction of cytochrome c. Three functions of M Φ s from diabetic ODUS/Odu were significantly less than those of M Φ s from non-diabetic ODUS/Odu, diabetic or non-diabetic control rats. These suggest that diabetes strongly affects the leukocyte function of M Φ , thereby weakening the host defense mechanism, and leading to production of severe periodontal lesions. (Supported by a Grant-in-Aid for Scientific Research No.04671147)

P10.5.6

THE POSSIBLE CONTRIBUTION OF NEUTROPHILS, BUT NOT T LYMPHOCYTES, TO MDP-LYS(L18)-INDUCED ARTHRITIS IN RATS.

T. SUGAWARA, S. TAKADA, M. MIYAMOTO and M. KATO.
Drug Safety Research Center, Developmental Research Laboratories, Daiichi Pharmaceutical Co., Ltd., Tokyo, Japan.

When MDP-Lys(L18), an analogue of muramyl dipeptide, was subcutaneously administered to rats, swelling of the tarsal joints was seen from 1 week later. We speculate that MDP-Lys(L18)-induced arthritis (MIA) is different from Freund's complete adjuvant-induced arthritis (AIA). In the present study, we investigated the contribution of T lymphocytes to the development of MIA and AIA. AIA was not induced in athymic nude rats, and spleen cells obtained from Lewis rats with AIA transferred the arthritis to normal recipients. In contrast, MIA was induced in nude rats and was not transferred by spleen cells. We also examined the contribution of neutrophils to MIA because neutrophil infiltration was the earliest change observed in synovial membrane. MDP-Lys(L18) stimulated the production of neutrophil chemotactic factor(s) by rats peritoneal macrophages *in vitro*, but did not by those of mouse which never showed MIA. These data suggest that neutrophils recruited by chemoattractants released from MDP-Lys(L18)-activated macrophages may have an important role in MIA.

EFFECT OF BIO-NORMALIZER ON MACROPHAGE CHEMOTAXIS IN NATURALLY OCCURING GINGIVITIS RAT

M. Shinohara, Y. Morikawa, K. Ogata, J. A. Osato¹ and K. Ohura

Osaka Dental University, Osaka, Japan and ¹Osato Research Institute, Gifu, Japan

(Poster paper presented at the 12th International Congress of Pharmacology, 24 ~ 29
July 1994, Montreal, Canada)

This study examined the effect of two- month administration of Bio-normalizer (BN) on the chemotaxis of macrophages in naturally occurring gingivitis rat (ODUS/Odu) injected with 1% oyster glycogen solution. BN significantly decreased the macrophage chemotaxis as compared to the control group of rats suggesting that BN may play an immunological role in host defense.

XIIth International Congress of Pharmacology

Program

Molecular Mechanisms to
Modern Medicine

XII^e Congrès international de pharmacologie

Programme

Des mécanismes moléculaires
à la médecine moderne

表紙見本

本から カラーコピー

して下さい



Montréal, Canada
July 24-29 juillet 1994

A-①

XIIth International Congress of Pharmacology

PROGRAM

Molecular Mechanisms to Modern Medicine

Sponsored by

International Union of Pharmacology*
Pharmacological Society of Canada
Society of Toxicology of Canada
Canadian Society of Clinical Pharmacology
National Research Council Canada

*Member of the International Council of Scientific Unions

Congress Secretariat

Mail all correspondence to:
Nicole A. Sarault, Congress Manager
XIIth International Congress of Pharmacology
National Research Council Canada
Bldg. M-19, Montréal Road
Ottawa, ON, Canada K1A 0R6

XII^e Congrès international de pharmacologie

PROGRAMME

Des mécanismes moléculaires à la médecine moderne

Parrainé par

l'Union internationale de pharmacologie*
la Société de pharmacologie du Canada
la Société de toxicologie du Canada
la Société canadienne de pharmacologie clinique
le Conseil national de recherches Canada

*Membre du Conseil international des unions scientifiques

Secrétariat du Congrès

Adresser la correspondance à :
Nicole A. Sarault, Gestionnaire du congrès
XII^e Congrès international de pharmacologie
Conseil national de recherches Canada
Édifice M-19, Chemin de Montréal
Ottawa, ON, Canada K1A 0R6



Montréal, Canada
July 24-29 juillet 1994

コピー
原稿

- P10.4.33 (368) NaCl HYPERTONIC SOLUTION PARTIALLY BLOCKS VASCULAR PERMEABILITY INDUCED BY MEDIATORS OF INFLAMMATORY PROCESS IN RATS, **M.N.C. Abel**, B.E. Malucelli, *University of São Paulo, Brazil*
- P10.4.34 (369) SYNERGISTIC EFFECT OF VASOACTIVE AMINES ACCOUNTS FOR ANTIGEN-INDUCED PLEURAL LEAKAGE IN ACTIVELY IMMUNIZED RATS, **M.C.R. Lima**, M.S.S. Chagas, P.M.R. e Silva, H.C. Castro-Faria-Neto, R.S.B. Cordeiro, M.A. Martins, *Oswaldo Cruz Institute, Rio de Janeiro, Brazil*
- P10.4.35 (370) DIFFERENTIAL CELLULAR ACCUMULATION TRIGGERED BY PAF IN THE PERITONEAL CAVITY OF SENSITISED MICE, **C. Zuany-Amorim**¹, D.C. Deslandes¹, A.P. Hofer¹, R.S.B. Cordeiro¹, M. Pretolani¹, B.B. Vargaftig², ¹IOC/FIOCRUZ, *Rio de Janeiro, Brazil*; ²Institut Pasteur, *Paris, France*
- P10.4.36 (371) COMPARISON OF NON-PEPTIDE NK-1 RECEPTOR ANTAGONISTS AND CALCIUM CHANNEL ANTAGONISTS ON THE INHIBITION OF THE PLASMA EXTRAVASATION INDUCED BY THERMAL INJURY IN RATS, **G.M. Pitcher**, J.L. Henry, T.J. Coderre, *Clinical Research Institute of Montréal and McGill University, Montréal, QC, Canada*
- P10.4.37 (372) CONTROL OF RAT MAST CELL ACTIVITY THROUGH HISTAMINE H₃-RECEPTOR, **V. Dimitriadou**¹, M. Garbarg², A. Rouleau², M. Dam Trung Tuong², G. Newlands³, J.-C. Schwartz¹, ¹Université de Paris V, *Paris* and ²INSERM U-109, *Paris, France*; ³Moredun Institute, *Edinburgh, UK*
- P10.4.38 (373) THE ROLE OF NEUROPEPTIDES IN THE PATHOGENESIS OF CUMULATIVE TRAUMA DISORDERS, **R. Kijowski**, D. Hoppensteadt, M. Chinthagada, G. Chejfec, J. Fareed, S. Blair, *Loyola University, Maywood, IL, USA*
- P10.4.39 (374) GASTRODUODENAL MUCOSA PAF LEVELS BEFORE AND AFTER TREATMENT OF HELICOBACTER PYLORI INFECTION, **V.D. Pasechnikov**, N.V. Zhurbina, I.V. Kuznetsova, *Stavropol Medical Institute, Stavropol, Russia*

Inflammation: Other

P10.5

Autres inflammation

- P10.5.1 (375) GENERATION OF LEUKOTRIENE C₄ AND HISTAMINE RELEASE WITH PHOSPHATIDIC ACID VIA CALCIUM INFLUX IN MASTOCYTOMA CELL LINE, **Y. Moriya**, H. Teshigawara, *Nihon University School of Dentistry at Matsudo, Chiba, Japan*
- P10.5.2 (376) EFFECTS OF LPS AND INDOMETHACIN ON SHORT TERM FEVER RESPONSE AND HYPOTHALAMIC PGE₂ PRODUCTION IN MICE, **L. Abramovich**, V. Fraifeld, D. Shemi, J. Kaplanski, *Ben Gurion University of the Negev, Beer Sheva, Israel*
- P10.5.3 (377) EFFECT OF BIO-NORMALIZER ON MACROPHAGE CHEMOTAXIS IN NATURALLY OCCURRING GINGIVITIS RAT, **M. Shinohara**¹, Y. Morikawa¹, K. Ogata¹, A.J. Osato², K. Ohura¹, ¹Osaka Dental University, *Osaka* and ²Osato Research Institute, *Gifu, Japan*

コゼー原稿

- Okagaki, T. P21.1.22(A)
 P21.1.23(A)
 Okamoto, H. P19.1.14
 Okamoto, K. P13.2.14
 Okamoto, M. P13.9.23
 Okamoto, T. P13.14.8
 Okano, Y. P12.2.6
 Okawara, H. P1.9.30(A)
 Okazaki, K. P1.4.25
 Okazaki, T. P1.8.11
 Okazoe, T. P1.9.34(A)
 Oker-Blom, C. P13.1.12
 Okey, A.B. S4.3
 Oki, H. P18.13.11
 Okinaga, M. P13.6.11
 Okinaga, S. P24.1.18(B)
 Okita, J.R. P12.1.7
 Okita, R.T. P12.1.7
 Okonkwo, P.O. P2.1.37
 Okoye, Z.S.C. P22.2.34
 Okoyeh, J.N. P2.1.47
 Oksala, O. P1.15.13
 Oktay, S. P13.9.33
 Okubo, M. P18.6.1
 Okuda, H. P13.9.23
 Okuda, K.-I. S16.5
 Okuhara, M. P5.1.10
 Okujava, N.V. P13.13.28
 Okujava, V.M. P13.13.27
 Okulicz-Kozaryn, I. P13.5.36
 P13.15.15
 Okuma, Y. P13.19.40
 Okumura, Y. P1.8.6
 Okuniewski, R. P1.15.45
 Olah, T.V. P12.1.33
 Olejnik, A. P1.9.59(B)
 P1.9.78(B)
 Olenina, N.G. P1.1.32
 Olinas, M.C. P13.5.22
 P18.9.13
 Oliva, L. P13.26.14
 Oliva, M.L.V. P14.4.1
 Olivato, P.R. P2.2.2
 Oliveira, A.C. P13.9.20
 Oliveira, B. P10.1.2
 Oliveira, L. P18.9.22
 Oliveira, M.J. P13.9.20
 Oliver, D.W. P18.11.1
 P18.13.9
 Olkkola, K.T. P12.2.2
 P12.2.27
 Olsen, R.W. P18.4.7
 S28.2
 Olson, L. S10.4
 Olsson, R.A. P17.2.3
 P17.3.11
 Olsson, T. P9.1.14
 Olubadewo, J.O. P1.9.86(B)
 P22.3.32
 Olverman, H.J. P17.2.17
 Olymulder, C.G. P16.2.19
 Omar, R.F. P12.1.10
 Omer, S. P19.1.8
 Omogbai, E.K.I. P1.13.91(B)
 S49.4
 Omori, K. P8.1.4
 P13.13.7; P13.26.4
 Omri, A. P2.1.33
 Omura, H. P24.1.18(B)
 Onali, P. P13.5.22
 P18.9.13
 Onat, F. P13.9.33
 Ondeyka, D. P1.9.65(B)
 P1.15.19
 Öner, U. P22.1.37
 Ong, H. P12.2.21
 P13.17.23; P18.12.6
 Ono, K. P1.6.11
 P18.5.17
 Ono, N. P13.8.14
 Ono, S. P12.1.5
 Ono, T. P1.10.9
 Ono, Y. P5.2.32
 Onodera, K. P13.14.5
 P13.14.11
 Onomo, J.P. P13.18.11
 Onur, R. P1.13.92(B)
 P20.1.11
 Ooi, Y. P13.19.30
 Oortgiesen, M. P9.1.54
 Oosting, J. P1.9.91(B)
 P23.1.16
 Oparin, D.A. P12.1.46
 Opezzo, J. P19.1.25
 Oppen, C. P2.1.29
 Oppermann, U.C.T. P12.3.45
 Oprea, T.I. P4.1.20
 Orallo, F. P1.15.31
 P14.2.15; P14.3.13
 Orbo, A. P18.2.1
 P18.2.2
 Orekhova, E. P4.1.43
 Orhii, P.B. P5.2.30
 Orikasa, M. P22.1.11
 Oriowo, M.A. P13.1.24
 Orita, A. P6.2.16
 Orita, Y. P19.1.30
 Orlov, L. P1.9.101(B)
 Orlov, S.N. P18.2.16
 Ormazabal, M.J. P13.4.7
 P13.4.25; P13.5.53
 Orme, M. P24.1.11(A)
 Ormsby, E. P22.1.27
 Orrenius, S. S40.4
 Ortega, M.P. P13.20.29
 Ortin, F. P22.2.19
 Ortiz de Montellano, P.R. PL18
 Ortiz, J.A. P6.3.9
 P13.16.1
 Ortiz, J.G. P13.2.27
 Ortiz, J.L. P16.1.7
 Orvig, C. P5.2.14
 Osato, A.J. P10.5.3
 Oshiro, M.E.M. P21.1.26(A)
 Oshita, M. P13.1.6
 Osinski, M.A. P6.1.2
 Osman, N. P1.13.106(B)
 Oster, L. P1.9.67(B)
 P1.9.69(B); P1.15.4
 Ostrovskaya, R.U. P4.1.38
 P13.17.46
 Osugi, T. P13.12.6
 Osuma, Y. P13.19.40
 Otabekova, S. P12.2.66
 Otani, H. P13.17.20
 Otieno, R.A. P22.3.32
 Otsuguro, K. P13.19.41
 Otsuka, M. PL9
 Ott, T. P13.17.39
 Otterness, D. P12.3.3
 Otterness, I. P10.1.47
 Ottobrelli, A. P12.2.20
 Ou, R.-C. P1.9.83(B)
 Oudart, N. P14.4.5
 Ouellet, M. P10.1.19
 P10.1.21; P10.3.10
 P10.4.29
 Ouerfelli, O. P2.2.32
 Ouimet, N. P18.9.33
 Ounissi, H. P10.1.46
 Ouseph, A.K. P10.3.15
 P18.9.50
 Ouvina, G. P12.1.38
 Overfield, J. P11.1.1
 Oviedo, C. P5.1.12
 Ovsiannikov, V.I. P13.1.33
 Owen, A. P4.1.25
 Owen, C. P4.1.16
 Owen, M. P1.13.13(A)
 Owicki, J.C. P13.9.32
 P23.1.11
 Owolabi, J. P13.11.11
 Oxender, D.L. P13.25.6
 Oyamada, M. P18.13.15
 Ozaki, H. P14.4.14
 Ozaki, S. P1.14.33
 Ozaki, Sy. P1.9.36(A)
 Ozawa, S. P12.3.5
 S35.3
 Özdemir, M. P19.1.29
 Ozkutlu, U. P13.9.33
 Paakkari, I. P1.5.21
 P1.9.62(B); P13.5.35
 Paakkari, P. P13.5.35
 Pabla, R. P1.11.1
 Pacak, K. P12.2.43
 Pacheco, M.A. P13.11.21
 Pacheco, O. P10.1.58
 Padiou, P. P5.2.17
 Padua, R. P13.18.10
 Paeile, C. P13.4.21
 Paetsch, P.R. P7.1.7
 Pagé, B. P9.1.58
 Page, C. P2.1.8
 Page, C.P. P1.10.21
 P9.1.32; P16.2.28
 P16.2.29; P16.2.30
 P16.2.31