

# **Fourth Vienna Shock Forum in Association With the European Shock Society**

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# Author-Abstract Index\*

- Aasen, A.O., 10.4, 12.36P  
Albrecht, D.M., 12.29P, 12.40P  
Albrecht, M., 11.13P  
Altavilla, D., 7.5  
Ammann, D., 12.51P  
Ammons, W.S., 12.5  
Åneman, Anders, 6.11P  
Angehrn, Peter, 12.10  
Appel, R., 5.4, 5.7P, 11.2  
Arnestad, J.P., 4.20P  
Assfalg-Machleidt, I., 12.37P  
Athwal, Dee, 12.52  
Ayala, A., 4.1, 4.5
- Bach, F., 7.8P  
Bahrami, S., 4.12P, 4.13P, 7.3, 12.11, 12.25P, 12.33P  
Baier, J., 4.16P  
Balassa, A., 1.6, 2.3  
Ball, Elaine, 7.6  
Banner, David, 12.10  
Bantel, C., 12.43P  
Barclay, R., 12.3  
Bardenheuer, M., 11.4  
Bartels, H., 5.9P  
Barthlen, W., 5.9P  
Baue, Arthur E., 6.2  
Bauer, M., 7.3, 7.8P, 11.11  
Becker, Heinz, 4.19P  
Becker, P., 5.8P  
Beger, H.G., 10.7  
Beloborodov, Vladimir B., 5.14P, 10.14P  
Bemelmans, M.H.A., 10.5, 10.29P  
Bengtson, J.P., 4.20P  
Bengtsson, A., 4.20P, 10.3  
Berger, A., 5.12P  
Berger, D., 1.5  
Berger, Hans-Jörg, 9.16P  
Berling, R., 5.16P  
Binnewies, C., 12.26P  
Blum, W.F., 11.10  
Bodmer, Mark, 12.52  
Boekstegers, P., 8.7, 9.2, 11.7  
Boldrini, G., 10.24P
- Bone, Roger C., 5.1  
Bonta, Il., 8.12P  
Booy, L.D.H.J., 4.21P  
Bordoli, M., 8.10P  
Boros, M., 6.7P, 6.9P  
Borowa, O., 11.19P  
Brade, H., 12.3, 12.24P  
Brain, J.D., 12.13, 12.34P  
Brandtzaeg, P., 10.3  
Brankova, J., 4.17P  
Braun, G., 10.2, 10.19P, 12.13  
Brear, S.G., 4.3  
Brenchley, P.E.C., 4.3  
Brinker, Jens, 4.19P  
Brofeldt, B.T., 12.19  
Browning, J., 10.8  
Bruch, H.P., 12.27P  
Bruch, J., 10.17P  
Büchler, M., 10.7  
Bühren, V., 7.3, 11.5, 11.11, 12.51P  
Bujdoso, O., 5.7P  
Burchardi, H., 12.15  
Burns, H.O., 8.6  
Buurman, W.A., 7.4, 10.5, 10.29P  
Bux, R., 1.5
- Campo, G.M., 7.5  
Canale, P., 7.5  
Caputi, A.P., 7.5  
Cardoza, T.A., 4.6, 10.10P  
Carlsen, H., 10.4, 12.36P  
Carnathan, G.W., 12.23  
Carpenter, A., 8.9P  
Cassuto, J., 12.20  
Castagneto, M., 10.23P  
Chaplin, Lesley, 12.52  
Chaudry, I.H., 4.1, 4.5  
Chen, Miin-Fu, 10.15P  
Cheronis, J.C., 12.23  
Chiarla, C., 10.23P, 10.24P  
Chrétien, J.M., 11.6  
Clements, Gary, 7.6  
Cohen, J., 8.2, 8.9P  
Cox, C.S., 12.49P  
Czech, Jörg, 12.39P
- Czech, T., 2.4
- D'Agostino, Horacio, 9.7  
d'Arcy, Alan, 12.10  
Dandona, P., 9.5  
Dang, Y., 10.16P  
Dapper, F., 11.21P  
Dauberschmidt, R., 5.15P, 10.25P  
Davies, J., 7.4, 12.9  
De Baetselier, Patrick, 7.6  
De Groote, D., 10.6  
Deitch, Edwin A., 6.3  
Demers, B., 5.17P  
Dhainaut, J.F., 11.6, 11.8  
Di Padova, F.E., 12.3  
Dickneite, Gerhard, 12.39P  
Dike, J., 11.5, 12.51P  
DiPadova, F., 12.24P  
Donker, A.J.M., 9.17P  
Dresing, K., 10.17P, 11.4  
Dreyfuss, D., 11.6  
Duncker, H.P., 6.6, 10.18P, 12.7, 12.44P  
Duncker, H.R., 10.18P  
Durham, R.M., 9.6  
Dwenger, A., 2.2, 4.9, 11.16P, 12.43P  
Dzeksenbaev, Oleg Sh., 5.14P, 10.14P
- Ehlers, M., 12.2  
Eisold, C., 11.4  
Ellerbeck, M., 2.2  
Ellinger, K., 11.13P  
Emerson, T., 12.8  
Emtage, Spencer, 12.52  
Endo, S., 4.10P, 11.17P  
Enssle, Karlheinz, 12.39P  
Entman, M.L., 7.2  
Ertel, W., 4.4, 4.5, 10.11P, 12.35P  
Esch, Oliver, 9.7, 12.14  
Evans, T.J., 8.9P  
Eymann, J., 12.35P
- Fagerhol, M., 6.8P  
Fähnle, M., 11.13P  
Faist, E., 4.4, 4.15P, 10.11P, 12.35P  
Falkenhagen, D., 12.28P

\*Numbers correspond to abstract number, not page number.

- Farthmann, E.H., 4.14P  
 Fatehi-Hassanabad, Z., 8.5  
 Fattori, L., 8.10P  
 Ferguson, K., 5.17P  
 Feuerstein, Giora, 12.48P  
 Fisher, C.J., 5.2, 11.8  
 Foëx, B.A., 4.3  
 Fosse, E., 10.3  
 Fournel, M., 12.8  
 Frank, G., 12.12  
 Fritsch, A., 5.11P  
 Fritz, H., 11.3, 12.12  
 Froon, Albert H.M., 10.29P  
 Függer, R., 5.10P, 5.11P  
 Fundell, Larry, 9.7  
 Furman, B.L., 8.5, 8.6  
 Fürst, W., 9.3, 9.12P  
  
 Gallati, H., 4.3  
 Gardinali, M., 8.10P  
 Garvik, L.J., 6.8P  
 Gasser, H., 10.21P  
 Gegner, J., 12.1  
 Gehr, Gisela, 12.10  
 Gelderen, M.v., 8.12P  
 Gentz, Reiner, 12.10  
 Germann, P., 1.6, 2.3  
 Gimbrere, J.S.F., 4.21P  
 Giovannini, I., 10.23P, 10.24P  
 Gippner-Steppert, C., 12.16  
 Goldmann, K., 12.15  
 Gonschorek, O., 1.5, 1.8P  
 Göransson, J., 5.16P, 5.18P  
 Goris, R.J.A., 6.5, 10.20P  
 Götze, O., 12.15  
 Götzinger, P., 5.10P, 5.11P  
 Gram, H., 12.3  
 Greger, C., 9.12P  
 Greve, Jan Willem, 10.29P  
 Grob, P., 9.10P  
 Grochowsky, T., 11.19P  
 Grochowsky, W., 11.19P  
 Groeneveld, A.B.J., 9.8  
 Gross-Weege, Wilhelm, 4.19P  
 Grotz, M., 3.5, 11.16P  
 Grouix, B., 5.17P  
 Grüneberg, H., 5.15P  
 Grunwald, U., 12.2  
 Guinot, Ph., 11.6  
 Gunther, R.A., 12.19  
 Gustafsson, T., 12.19  
  
 Haase, E., 12.42P  
 Haglind, Eva, 6.11P  
 Haglund, Ulf, 6.4  
  
 Haller, I., 12.9  
 Hallström, S., 9.3, 9.12P, 9.14P  
 Halperin, José A., 9.16P  
 Hamar, J., 6.10P  
 Hamid, L.W., 5.13P  
 Hammerle, A., 11.10  
 Han, J., 12.1  
 Harrell, F., 5.2  
 Hartmann, Th., 1.6, 2.3  
 Hasibeder, W., 1.4, 10.22P  
 Hayes, M.A., 8.11P, 9.4, 11.9  
 He, Chang, 9.11P, 9.15P  
 He, Jie, 8.13P  
 Hedwig-Geissing, M., 10.2, 10.19P  
 Heemskerk, A.E.J., 9.17P  
 Hehrlein, F., 11.21P  
 Hellinger, A., 1.7P  
 Hendriks, Th., 4.21P, 6.5, 10.20P  
 Herold, M., 4.17P, 10.22P  
 Hieronymi, U., 10.25P  
 Higgs, Gerry, 7.6  
 Hillegas, Leonard, 12.48P  
 Hinds, C.J., 8.11P, 9.4, 11.9  
 Hobbensiefken, G., 2.2  
 Hochleuthner, Hannes, 11.12  
 Hofer, H.P., 3.7P  
 Hoffmann, G., 10.7  
 Hoffmann, H., 12.12  
 Høgåsen, K., 10.3  
 Holler, E., 11.7  
 Holwegler, Ch., 4.14P  
 Holzapfel, L., 11.6  
 Hölzenbein, Th., 11.10  
 Homann, H-H., 4.16P, 11.15P  
 Hopf, R., 5.19P  
 Höpken, U., 12.15  
 Hoshi, S., 4.10P, 11.17P  
 Howat, David, 7.6  
 Hoyt, D.B., 4.6, 10.10P  
 Hu, Sen, 10.28P  
 Huang, Zhi-hong, 10.13P  
 Huemer, G., 2.4  
 Hughes, B.J., 7.2  
 Hughes, Bernadette, 7.6  
 Huisman, E., 9.17P  
 Hültner, L., 4.4, 4.15P  
 Hwang, Tsann-Long, 10.15P  
  
 Ickert, K., 5.15P  
 Illyés, 6.10P  
 Imhof, H.-G., 9.10P  
 Inada, K., 4.10P, 11.17P  
 Inthorn, D., 11.3  
 Ioculano, M., 7.5  
 Irle, K., 11.21P  
  
 Ishizaka, A., 12.38P  
 Issels, Rolf, 9.12P  
 Istfan, N., 12.45P  
 Izbicki, J.R., 12.14  
 Izzo, J.L., Jr., 9.5  
  
 Janíček, A., 4.2  
 Jarrar, D., 10.11P  
 Jeki, I.M., 11.18P  
 Jeki, M. Lj., 11.18P  
 Jelinková, M., 4.2  
 Jesmok, G., 12.8  
 Jiang, J.X., 4.12P, 4.13P, 12.25P  
 Jochum, M., 3.4, 11.3, 11.4  
 Joller, H., 9.10P  
 Jönsson, A., 12.20  
 Jönsson, K., 5.16P  
 Juhász, M., 6.10P  
 Junger, W.G., 4.6, 10.10P  
  
 Käab, S., 9.2, 11.2  
 Kaminski, D.L., 9.6  
 Kanazawa, M., 12.38P  
 Kantorová, I., 4.2, 11.20P  
 Karner, J., 11.10  
 Karres, I., 12.35P  
 Kasai, T., 1.17P, 4.10P  
 Kaszaki, J., 6.7P, 6.9P  
 Katz, A., 10.8  
 Kaukemüller, J., 9.9  
 Kaul, M., 11.7  
 Kawashiro, T., 12.38P  
 Kazandzjev, G., 4.17P  
 Kemen, M., 4.16P, 11.15P  
 Kempeni, J., 11.7  
 Kenney, J., 10.11P  
 Keser, C., 12.16, 12.37P  
 Kinzl, L., 1.5, 1.8P  
 Kirkland, T., 12.1  
 Kirkman, E., 2.5  
 Kirste, G., 4.14P  
 Klein, A., 12.44P  
 Klein, B., 10.17P  
 Kleinschmidt, Chr., 1.3, 10.17P, 11.4  
 Klinkmann, H., 12.28P  
 Kloppel, T.M., 12.23  
 Klosterhalfen, B., 4.18P, 7.9P  
 Knaus, W.A., 5.2  
 Kneidinger, R., 7.4  
 Koch, T., 6.6, 10.18P, 12.7, 12.44P  
 Kohn, F., 12.5  
 Koidl, B., 9.3  
 Kolomiytsev, V., 10.27P, 12.50P  
 Konerding, M.A., 1.7P  
 Kossmann, T., 3.2, 9.10P

- Kotzerke, J., 4.9  
 Kowasky, R., 11.19P  
 Kramer, G.C., 12.49P  
 Kramer, L., 5.6P  
 Kranz, A., 5.6P  
 Kravchenko, V., 12.1  
 Kremer, J.-P., 4.4, 4.15P, 12.35P  
 Kreuzer, E., 11.2  
 Krieter, H., 12.40P  
 Krönke, M., 5.9P  
 Kropp, E., 12.8  
 Krösl, P., 5.19P  
 Kroworsch, P., 12.16, 12.37P  
 Krüger, C., 12.2  
 Krumm, K., 12.43P  
 Kuckelt, W., 10.25P  
 Kühn, E., 12.13  
 Kukovetz, E., 3.7P  
 Kung, A.H.C., 12.5  
 Kürten, K., 12.46P  
 Kurzweil, S., 9.6  
 Kuschinsky, W., 12.29P  
  
 Lachmann, B., 12.21  
 Lacquet, L.K., 4.21P  
 Lalani, S., 4.7  
 Lamb, W.R., 4.3  
 Larsen, R., 7.8P, 11.11  
 Lasson, Å., 5.16P, 5.18P  
 Lau, Ying-Tung, 10.15P  
 Lavergne, V., 11.6  
 Leach, W., 12.5  
 Lee, J.-D., 12.1  
 Lehmann, U., 2.2, 3.5, 11.16P  
 Lehmkuhl, P., 9.9  
 Lehn, N., 5.9P  
 Leichtfried, G., 4.12P, 4.13P, 12.25P,  
 12.33P  
 Lenz, K., 5.6P  
 Lesslauer, Werner, 12.10  
 Letulzo, Y., 11.6  
 Leturcq, D., 7.7, 12.1  
 Liehl, E., 12.3  
 Lille, Yu. E., 12.41P  
 Linder, L., 11.21P  
 Little, R.A., 2.5  
 Liu, F.C., 4.6, 10.10P  
 Liu, Maw-Shung, 10.15P  
 Loetscher, Hansruedi, 12.10  
 Löffler, Th., 12.14  
 Loomis, W.H., 10.10P  
 Lopp, M.J., 12.41P  
 Loth, F., 12.28P  
 Low, D., 5.17P  
 Lowry, Stephen, 12.10  
  
 Luchtefeld, W.B., 9.6  
 Lund, Tjøstolv, 12.18  
 Lutz, F., 12.7  
 Lyberg, T., 6.8P  
  
 Macartney, I.D., 4.3  
 Machleidt, W., 11.3, 12.37P  
 Mackay, Fabienne, 12.10  
 Mackway-Jones, K., 2.5  
 Madl, C., 5.6P  
 Maghsudi, M., 3.3  
 Malik, Asrar B., 7.1  
 Malkusch, W., 1.7P  
 Mallari, C., 12.5  
 Mann, D., 2.2  
 Mariscalco, M.M., 7.2  
 Markewitz, A., 4.4  
 Marzi, I., 7.3, 7.8P, 11.11  
 Mathes, C., 12.15  
 Mathison, J.C., 12.1  
 Matsuura, Nariaki, 12.31P, 12.32P  
 Mauritz, W., 5.13P, 11.12  
 Mayer, G., 11.21P  
 Mazuski, J.E., 9.6  
 McGeer, A., 5.17P  
 McGinn, P., 5.4, 5.7P, 9.2  
 McGinn, P.V., 11.14P  
 Medbak, Sami, 6.11P  
 Meier, E., 12.7  
 Menke, A., 5.12P  
 Meyer, J., 12.49P  
 Meyer, M., 10.25P  
 Mikol, V., 12.24P  
 Minion, Tina, 7.6  
 Mira, J.P., 11.6  
 Mitchell, J.A., 8.3, 8.4  
 Mitzner, S., 12.28P  
 Mladenov, M., 4.17P  
 Mohr, M., 12.15  
 Moldawer, Lyle L., 12.10  
 Mollnes, T.E., 10.3  
 Montz, H., 12.15  
 Morganti-Kossmann, M.C., 9.10P  
 Mori, Takesada, 12.31P  
 Moriarty, A., 7.7, 12.1  
 Morrison, M.H., 4.5  
 Mozes, T., 8.12P  
 Mrochen, H., 5.15P, 10.25P  
 Mühlbacher, F., 5.10P  
 Müller, U., 9.3, 9.11P, 9.12P, 9.15P  
 Müller-Höcker, J., 10.9  
 Mumme, A., 11.15P  
 Murata, Atsuo, 12.31P, 12.32P  
 Mutschler, W., 11.5  
 Mutz, N.J., 1.4, 10.22P  
  
 Naess, F., 10.4  
 Nagy, S., 6.7P, 6.9P  
 Nakamura, H., 12.38P  
 Nakazawa, H., 12.17, 12.49P  
 Næss, O., 6.8P  
 Nast-Kolb, D., 3.4  
 Nebermann, L., 12.6  
 Nellgård, P., 12.20  
 Nerlich, M., 3.3  
 Nespoli, A., 8.10P  
 Neudeck, F., 11.4  
 Neuhof, Ch., 11.21P  
 Neuhof, H., 6.6, 10.18P, 11.21P,  
 12.7, 12.44P  
 Neumann, C., 3.3  
 Nitsche, D., 8.8, 12.6, 12.26P  
 Nix, D.E., 9.5  
 Nowak, M.C., 4.7  
  
 O'Laoide, Ristead, 9.7  
 Obertacke, U., 1.3, 1.7P, 10.17P, 11.4  
 Oberthür, Walter, 9.15P  
 Oborin, A.N., 10.26P, 12.41P, 12.47P  
 Ochmann, J., 11.20P  
 Oglevie, Steven, 9.7  
 Öhlinger, W., 7.4, 12.22  
 Olinger, A., 11.5  
 Omeljanenko, O., 12.50P  
 Opal, S., 5.2  
 Opal, S.M., 11.8, 12.4  
 Oppermann, M., 12.15  
 Ördögh, B., 6.7P, 6.9P  
 Ostertag, P., 12.14  
 Otto, V., 12.27P  
  
 Padalino, P., 8.10P  
 Palazzo, M., 11.9  
 Pallua, N., 5.12P  
 Pape, H.-C., 3.5, 4.9, 12.43P  
 Parratt, J.R., 8.1, 8.5, 8.6  
 Parrillo, Joseph E., 9.1  
 Passlick, B., 12.14  
 Paul, E., 11.11  
 Pavlovsky, M.P., 12.41P, 12.50P  
 Petek, W., 3.7P  
 Peters, W., 4.7  
 Petkov, J., 4.17P  
 Petricek, W., 1.6, 2.3  
 Pichlmayr, I., 9.9  
 Pilz, G., 5.3, 5.4, 5.7P, 9.2, 9.14P,  
 11.1, 11.2  
 Pilz, S., 4.15P  
 Pizanis, A., 11.5, 12.51P  
 Platonov, Alexander E., 5.14P,  
 10.14P

- Plochev, K., 4.17P  
 Pöschl, G., 1.6, 2.3  
 Poxton, I., 12.3  
 Pretorius, J.P., 5.8P  
 Pribble, J.P., 11.8  
 Pruzanski, W., 5.5P, 5.17P, 10.8  
 Pscheidl, E., 10.2, 10.19P, 12.45P  
 Pugin, J., 7.7, 12.1
- Rabinovici, Reuven, 12.48P  
 Rácz, I., 6.10P  
 Radoux, D., 10.6  
 Raffin, T.A., 12.12  
 Ravizzini, C., 8.10P  
 Redelmeier, D., 5.5P  
 Redl, H., 1.3, 1.7P, 3.6, 3.8P, 4.6,  
 4.12P, 4.13P, 4.20P, 7.3, 7.4,  
 9.11P, 10.3, 10.21P, 11.11,  
 11.16P, 12.9, 12.11, 12.22,  
 12.25P, 12.33P, 12.37P,  
 Regel, G., 2.2, 3.5, 4.9, 11.16P  
 Reichwein, A., 12.7  
 Reif, W., 2.2  
 Reisdorf, E., 7.3  
 Reithmann, C., 9.13P, 9.14P  
 Remick, D.G., 4.8  
 Riegel, W., 11.11  
 Rietschel, E. Th., 12.3  
 Rimbäck, G., 12.20  
 Roberts, T.E., 4.3  
 Robinson, M., 7.2, 7.4, 7.6  
 Rodell, T.C., 12.23  
 Rose, S., 11.5, 12.51P  
 Roth, E., 5.10P, 5.11P, 11.10  
 Rotheneder, E., 2.4  
 Roumen, R.M.H., 6.5, 10.20P  
 Roux, L.J., 5.8P  
 Rügheimer, E., 10.2, 10.19P, 12.45P  
 Rutter, Judy, 7.6
- Sadoff, J., 5.2  
 Sakamaki, F., 12.38P  
 Sanchez, Robert, 9.7  
 Sautner, Th., 5.10P, 5.11P  
 Saxena, Pr., 8.12P  
 Sayama, K., 12.38P  
 Sayeed, M.M., 12.51P  
 Schade, U., 12.40P  
 Schaur, R.J., 3.7P  
 Schedel, I., 5.12P  
 Schentag, J.J., 9.5  
 Schiefer, H.G., 6.6  
 Schildberg, F.W., 4.4, 10.11P, 12.35P  
 Schilling, T., 12.2  
 Schinkel, C., 4.4, 4.15P
- Schlaeger, Ernst-Juergen, 12.10  
 Schlag, G., 1.3, 1.7P, 3.6, 3.8P, 4.6,  
 4.12P, 4.13P, 4.20P, 5.13P,  
 5.19P, 6.1, 7.4, 9.3, 9.11P,  
 9.12P, 9.14P, 10.3, 10.21P,  
 11.11, 12.9, 12.11, 12.22,  
 12.25P, 12.33P  
 Schlemmer, B., 11.6  
 Schlichting, E., 6.8P  
 Schmidt, R., 12.28P  
 Schmit-Neuerburg, K.P., 1.3, 1.7P,  
 10.17P, 11.4  
 Schmoker, J., 2.1  
 Schneeweiss, B., 5.6P  
 Schneider, M., 10.12P  
 Schneidewind, J.M., 12.28P  
 Schnekenburger, E., 4.14P  
 Schöffel, U., 4.14P  
 Schouten, D., 5.5P  
 Schramm, W., 2.4  
 Schreiber, G., 5.15P  
 Schroth, M., 11.13P  
 Schulze, C., 8.8, 12.6, 12.26P  
 Schürer-Maly, C.C., 7.7  
 Schütt, Christine, 12.2  
 Schwartz, B., 5.17P  
 Schwarz, Ch., 10.22P  
 Schweiberer, L., 3.4, 12.14  
 Schweitzer, G., 11.16P, 12.43P  
 Schywalsky, M., 10.2, 10.19P, 12.45P  
 Scott, K., 10.8  
 Seekamp, A., 2.2, 3.5, 4.8  
 Segiet, W., 12.40P  
 Seifert, J., 12.42P  
 Semsroth, M., 11.10  
 Senkal, M., 4.16P, 11.15P  
 Seyr, M., 1.4, 10.22P  
 Shackford, S.R., 2.1  
 Shapiro, M.J., 1.1, 9.6  
 Shatos, M., 2.1  
 Sheng, Chih-yong, 10.13P  
 Sheng, Zhi-Yong, 4.11P, 10.28P,  
 12.30P  
 Sherman, D., 10.27P, 12.50P  
 Shi, Zhi-guo, 10.13P  
 Shock, Anthony, 7.6  
 Siebeck, M., 12.16, 12.37P  
 Siegel, J.H., 10.23P  
 Siever, S., 12.15  
 Siewert, J.R., 5.9P  
 Simon, P., 3.8P  
 Simon, S., 7.2  
 Simor, A., 5.17P  
 Slotman, G.J., 11.8  
 Smith, C. Wayne, 7.2
- Smith, Thomas W., 9.16P  
 Solet, J.P., 11.6  
 Song, Dekun, 8.13P  
 Sopwith, Mark, 12.52  
 Spannagl, E., 12.16, 12.37P  
 Spannagl, M., 12.12, 12.16  
 Specht, J., 1.3  
 Spies, B., 12.16, 12.37P  
 Spiss, C.K., 2.4  
 Squadrito, F., 7.5  
 Stange, J., 12.28P  
 Stark, M., 12.15  
 Staubach, K.H., 12.27P  
 Steinbach, G., 1.8P  
 Steinemann, S., 12.1  
 Steininger, R., 5.10P  
 Stelter, F., 12.2  
 Stephens, Sue, 12.52  
 Stieber, Ch., 12.40P  
 Stocker, R., 3.2, 9.10P  
 Stoclet, Jean-Claude, 8.1  
 Strecker, W., 1.5, 1.8P  
 Strohmaier, W., 1.3, 5.13P, 12.22  
 Stueber, Dietrich, 12.10  
 Sturm, J.A., 2.2, 11.16P  
 Stuwe, B., 12.42P  
 Sun, Shi-Rong, 4.11P, 10.13P, 12.30P  
 Sung, B.H., 9.5  
 Svoboda, P., 4.2, 11.20P  
 Symonds, W.T., 9.5  
 Szabó, A., 6.9P  
 Szabó, Cs., 8.3, 8.4
- Takkin, Ch., 4.14P  
 Tanaka, Nobuo, 12.31P, 12.32P  
 Tao, J., 10.16P  
 Tarnow, P., 12.20  
 Tasaka, S., 12.38P  
 Temple, Graham, 12.52  
 Tenailon, A., 11.6  
 Teodorczyk-Injeyan, J.A., 4.7  
 Terashima, T., 12.38P  
 Thiele, V., 10.11P  
 Thiemermann, C., 8.3, 8.4  
 Thijs, L.G., 9.17P  
 Thuma, B., 7.8P  
 Tian, Hui-Min, 4.11P, 12.30P  
 Till, G.O., 4.8  
 Timmins, A.C., 8.11P, 9.4, 11.9  
 Tobias, P.S., 7.7, 12.1  
 Toda, Hitoshi, 12.31P, 12.32P  
 Traber, Daniel L., 10.1, 12.17, 12.49P  
 Traber, L.D., 12.17, 12.49P  
 Trentz, O., 3.2, 9.10P

- Troshansky, Dimitry V., 5.14P, 10.14P  
 Tschaikowsky, K., 10.19P, 12.13, 12.34P  
 Tscherne, H., 3.5, 12.43P
- Uda, Kenichi, 12.31P, 12.32P  
 Uhl, W., 10.7  
 Ulevitch, R.J., 7.7, 12.1  
 Urano, T., 12.38P  
 Uspensky, B.A., 12.40P
- Vadas, P., 5.5P, 5.17P, 10.8  
 Valentini, L., 11.10  
 van Ackern, K., 6.6, 10.18P, 12.29P, 12.40P, 12.44P  
 van den Bos, G.C., 9.17P  
 Van der Linden, C.J., 4.21P, 10.29P  
 van Lambalgen, A.A., 9.17P  
 van Zee, Kimberley, 12.10  
 Van Zwienen, J.C.W., 4.21P  
 Vane, J.R., 8.4  
 Vane, R.J., 8.3  
 vanSonnenberg, Eric, 9.7  
 Varney, Manfred, 4.19P  
 Vernick, Jerome, 12.48P  
 Vetterlein, Olivia, 12.52  
 Viljoen, M., 5.8P  
 Vincent, J-L., 2.5  
 Vogel, P., 4.18P, 7.9P
- Vogeser, M., 10.7  
 Vogl, C., 9.3
- Waage, A., 10.4  
 Wagner, D.P., 5.2  
 Wagner, H., 5.9P  
 Waki, Y., 12.38P  
 Wang, X., 10.16P  
 Wang, Ya-Ping, 4.11P, 12.30P  
 Ward, P.A., 4.8  
 Warren, J.S., 4.8  
 Waschke, K., 12.29P  
 Wasner, M., 8.8  
 Watson, David, 6.11P  
 Watson, J.D., 8.11P, 9.4, 11.9  
 Waydhas, Ch., 3.4, 11.3  
 Weidenhöfer, S., 8.7, 9.2, 11.7  
 Weis, M., 12.16, 12.37P  
 Weiss, M., 10.12P  
 Werdan, K., 5.3, 5.4, 5.7P, 8.7, 9.2, 9.3, 9.11P, 9.12P, 9.13P, 9.14P, 9.15P, 11.1, 11.2, 11.7  
 Wernet, P., 10.12P  
 West, M.A., 12.23  
 Whalley, E.T., 12.23  
 Wilkinson, Michael, 7.6  
 Wilson, M.F., 9.5  
 Winkler, Fritz, 12.10  
 Winkler, S., 5.10P, 5.11P  
 Wolff, M., 11.6  
 Wozasek, G.E., 3.6, 3.8P
- Wu, Q., 10.16P
- Xu, Shi-Hao, 4.11P, 10.13P, 12.30P
- Yang, Qiongying, 8.13P  
 Yao, Yong-Ming, 4.11P, 12.30P  
 Yau, E., 8.11P, 9.4, 11.9  
 Yoshida, M., 4.10P, 11.17P  
 Yu, X., 10.16P  
 Yu, Y., 4.13P, 12.11, 12.25P  
 Yu, Yan, 4.11P, 12.30P
- Zadrobilek, E., 5.11P  
 Zauner, C., 5.6P  
 Zederfeldt, B., 5.16P  
 Zedler, S., 4.4, 10.11P  
 Zelnicek, P., 11.20P  
 Zeni, F., 11.6  
 Zeug, R., 1.8P  
 Zhang, H., 2.5  
 Zhang, Y., 10.16P  
 Zhao, Jun, 12.30P  
 Ziegenfuß, Th., 11.11  
 Ziegler-Heitbrock, H.W.L., 12.14  
 Zimmer, S., 4.15P, 4.4  
 Zimpfer, M., 1.6, 2.3  
 Zingarelli, B., 7.5  
 Znidar, S., 4.9  
 Zubachik, R.M., 12.41P  
 Zumtobel, V., 4.16P, 11.15P

#### 4.15P

##### *Transcriptional And Post-Translational Patterns Of Interleukin-6 (IL-6) Synthesis Following Massive Trauma And During Sepsis Syndrome*

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The objective of this study was to get further insight into the regulatory mechanisms of the inflammatory cytokine Interleukin-6 (IL-6) in patients following major injury or patients suffering from a manifest septic episode. Peripheral blood mononuclear cells (PBMC) were stimulated in-vitro by adherence, mitogen or endotoxin. The IL-6 protein release following stimulation was compared to the characteristics of the corresponding mRNA signal intensity in order to understand the regulatory levels of IL-6 synthesis under stressful conditions.

We prepared PBMC cultures from 9 polytraumatized patients (mean age  $33 \pm 3$  yrs., mean injury severity score (ISS)  $34 \pm 2$  pts) as well as from 10 patients ( $56 \pm 5$  yrs.) with postoperative sepsis. The PBMC's were collected on day 1,3,5,7 and 10 post-trauma or following the first clinical manifestation of a sepsis syndrome. After incubation with the different stimuli (adherence, lipopolysaccharide (LPS), phytohemagglutinin (PHA)), IL-6 protein was measured in the supernatants via a bioassay (celline 7TD1). Total RNA was isolated from the celllysates and IL-6 mRNA was detected after northern blotting via hybridization with the radiolabeled specific cDNA.

Despite heterogenous cell behaviour patterns of the individual patients, the results revealed that the cumulative in-vitro IL-6 synthesis

following trauma and during post-traumatic sepsis was significantly depressed compared to controls. A positive quantitative correlation between IL-6 protein release and IL-6 mRNA signal intensity was seen in  $> 80\%$  of cell samples studied.

From these results we conclude that the major regulatory factor, responsible for the stress induced inhibition of IL-6, is located on the transcriptional level. Mechanisms potentially responsible for this phenomenon, include inadequate activation of protein kinase C and hence resulting inhibition of the IL-6 gene via downregulation of the nuclear factor  $\kappa$ -B (NF $\kappa$ B), or other with the IL-6 promoter sequence interacting substances like cAMP.