GENTAUR

DATA SHEET

HIGHLY PURIFIED BACTERIAL LIPOPOLYSACCHARIDES AND RELATED PRODUCTS

Bacterial lipopolysaccharides have long been recognized as the active component of gram negative bacterial endotoxins.[1] These unique macromolecules have been extensively studied by investigators in many disciplines in efforts to elucidate and define relevant pathophysiological parameters of endotoxin shock, a profound life-threatening consequence of bacterial sepsis.[2] More recently these bacterial products have generated intense interest as being among the most potent natural products capable of pluropotential immunostimulatory activity. Such action can be manifested by the activation of host cells (e.g. B lymphocytes, macrophages) to functional differentiation.[3] In addition, however, host cell activation by lipopolysaccharides produces a spectrum of hormone-active lymphokines and monokines including the interferons (α, ß, γ), interleukins 1 and 6, tumor necrosis factor, platelet activating factor and procoagulant tissue factor.[4] Finally, the documented capacity of lipopolysaccharides or their active lipid A component to initiate a variety of bio- chemical pathways (protein kinase C,[5] cAMP dependent protein kinase,[6] phosphatidyl inositol turnover,[7] arachidonate metabolism,[8] protein myristolation[9] and activation of G-proteins[10]) provides investigators with powerful molecular tools by which to study cellular activation mechanisms.

Smooth strain lipopolysaccharides from *E. coli* and *S. typhimurium* are isolated by a modification of the phenol extraction method of Westphal and Jann.[11] Smooth strain lipopolysaccharides are dispersable in aqueous solvents at concentrations of up to 5.0 mg/ml. Rough strain lipopolysaccharides from *E. coli* and *S. minnesota* are isolated by a modification of the phenol-chloroform-petroleum ether extraction method of Galanos, *et al.*[12] and are dispersable at a concentration of 1.0 mg/ml in 0.5% triethylamine. All LPS preparations from Gentaur Molecular products are essentially free of nucleic acid and protein and are chemically characterized with respect to their phosphate and KDO (2-keto-3-deoxyoctonate) contents. Ultrapure LPS has been re-extracted by the method of Manthey and Vogel to eliminate residual protein contamination which may interfere with toll-like receptor studies.[13]

Gentaur Molecular products also offers two types of lipid A, a nontoxic form (primarily monophosphoryl) and a toxic version (primarily diphosphoryl). Monophosphoryl lipid A is prepared from *S. minnesota* R595 LPS by a modification of the method of Morrison and Leive[14] and typically contains less than 0.2% KDO. Diphosphoryl lipid A is prepared from *E. coli* K12, D31m4 LPS by a modification of the procedure of Takayama, *et al.*[15] and typically contains less than 0.1% KDO.

Each of the listed products are supplied as lyophilized powders. A detailed chemical analysis documenting purity accompanies each lot. **These products are intended for research purposes only and are not for use in humans**. For further information, please contact Gentaur Molecular products.

Ordering Information

Product No.	Lipopolysaccharides	Size
201	*Escherichia coli* 0111:B4	5.0 mg
203	*Escherichia coli* 055:B5	5.0 mg
206	*Escherichia coli* 0157:H7	10.0 mg
225	*Salmonella typhimurium*	5.0 mg
301	*Escherichia coli* J5 (Rc)	5.0 mg
302	*Escherichia coli* K12, D31m4 (Re)	5.0 mg
304	*Salmonella minnesota* R595 (Re)	5.0 mg
314	*Escherichia coli* K12, LCD25	1.0 mg
421	ULTRA PURE *Escherichia coli* 0111:B4	1.0 mg
423	ULTRA PURE *Escherichia coli* 055:B5	1.0 mg
434	ULTRA PURE *Salmonella minnesota* R595 (Re)	1.0 mg
	Related Compounds	
401	Lipid A (primarily monophosphoryl)	1.0 mg
402	Lipid A (primarily diphosphoryl)	1.0 mg

References

1. Westphal, O. and Lüderitz, O. (1954) *Agnew Chem.* **66**, 407-417.
2. Wolff, S.M. and Bennett, J.V. (1974) *N. Engl. J. Med.* **291**, 733-734.
3. Morrison, D.C. and Ryan, J.L. (1979) *Adv. Immunol.* **28**, 293-451.
4. Morrison, D.C. and Ryan, J.L. (1987) *Ann. Rev. Med.* **38**, 417-432.
5. Wightman, P.O. and Raetz, C.R.H. (1984) *J. Biol. Chem.* **259**, 10048-10052.
6. Suzuki, T. and Yamamoto, H. (1989) *FASEB J.* **3**, A1102.
7. Prpic, V., Weiel, J.E., Somers, S.D., DiGuiseppi, J., Gonias, S.L., Pizzo, S.V., Hamilton, T.A., Herman, B. and Adams, D.O. (1987) *J. Immunol.* **139**, 526-533.
8. Lüderitz, T. Brandenburg, K., Seydel, U. Roth, A., Galanos, C. and Rietschel, E.T. (1989) *Eur. J. Biochem.* **179**, 11-16.
9. Aderem, A.A., Keum, M.M., Pure, E. and Cohn, Z.A. (1986) *Proc. Natl. Acad. Sci.* **83**, 5817-5821.
10. Dziarski, R. (1989) *Eur. J. Immunol.* **19**, 125-130.
11. Westphal, O. and Jann, K. (1965) in R.L. Whistler (ed.) *Methods in Carbohydrate Chemistry* **Vol. 5**, pp. 83-91, Academic Press, New York.
12. Galanos, C., Lüderitz, O. and Westphal, O. (1969) *Eur. J. Biochem.* **9**, 245-249.
13. Manthey, C.L. and Vogel, S.N. (1994) *J. Endotoxin Research* **1,** 84-91.
14. Morrison, D.C. and Leive, L. (1975) *J. Biol. Chem.* **250**, 2911-2919.
15. Takayama, K., Ribi, E. and Cantrell, J.L. (1981) *Cancer Research* **41**, 2654-2657.