

2nd Fdition

Innate Immunity

Focus Toll-like Receptor (TLR) Agonists

The innate immune system plays an essential role in the host's first line of defense against microbial invasion and involves the recognition of pathogen-associated molecular patterns (PAMPs) or endogenous danger signals through the sensing of danger-associated molecular patterns (DAMPs) by pattern recognition receptors (PRRs). Activation of PRRs triggers cell signaling leading to the production of proinflammatory cytokines, chemokines and type 1 interferons, the induction of antimicrobial and inflammatory responses, pyroptotic cell death and the recruitment of phagocytic cells. These innate responses are responsible for efficient destruction and clearance of invading pathogens and other molecular threats and instructing the development of an appropriate pathogen-specific adaptive immune response.

The innate immune system comprises several classes of PRRs that allow the early detection of pathogens at the site of infection. The membrane-bound Toll-like receptors (TLRs) and C-type lectin receptors (CLRs) detect PAMPs in extracellular milieu and endosomal compartments. TLRs and CLRs cooperate with PRRs sensing the presence of cytosolic nucleic acids, like RNA-sensing RIG-I (retinoic acid-inducible gene I)-like receptors (RLRs; RLHs) or the DNA-sensing AIM2. Another set of intracellular sensing PRRs are the NOD-like receptors (NLRs; nucleotide-binding domain leucine-rich repeat containing receptors), which not only recognize PAMPs but also DAMPs. Upon stress (including infections and metabolic deregulation), certain NLRs form high molecular weight complexes called inflammasomes. These complexes and the self-degrading process autophagy play central roles in controlling innate and adaptive immunity (see Wallchart next Page).

Toll-like receptors (TLRs)

Toll-like receptors (TLRs) are a family of evolutionally conserved pattern recognition receptors (PRRs) expressed by a variety of cell types, particularly those of the innate immune system. TLRs are type I membrane glycoproteins, characterized by a cytoplasmic TIR (Toll/interleukin-1 receptor (IL- 1R)) domain and a leucine-rich repeat domain. They are capable of detecting exogenous **PAMPs** such as lipopolysaccharide (LPS), lipopeptides, flagellin, bacterial DNA and viral dsRNA, as well as endogenous, host-derived DAMPs, including HMGB1 and β -defensins.

The activation of TLR signaling pathways results in the production and release of various cytokines and chemokines. TLRs play a crucial role in host defence and inflammation and are implicated in the pathogenesis of immune diseases and cancer. TLR agonists are being tested as vaccines, enhancing tumor immunity by targeting immune checkpoints or inducing the expansion of T cells by potent adjuvants. Therefore, TLR agonists can activate both the innate and adaptive immune systems, play an important role in antiviral and antitumor immunity and are exploited as potent adjuvants to enhance tumor immunity.

SELECTED REVIEW ARTICLES The Role of Toll-Like Receptor in Inflammation and Tumor Immunity: X. Cen, et al.; Front. Pharmacol. **9**, 878 (2018) • Toll-like receptors: Activation, signalling and transcriptional modulation: D. De Nardo; Cytokine **74**, 181 (2015) • Characterization of innate immune signalings stimulated by ligands for pattern recognition receptors: T. Kameyama & A. Takaoka; Methods Mol. Biol. **1142**, 19 (2014)

www.adipogen.com

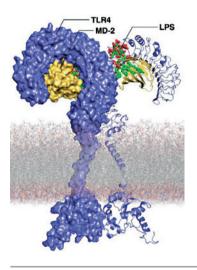


FIGURE 1: TLR4/MD-2/LPS complex.

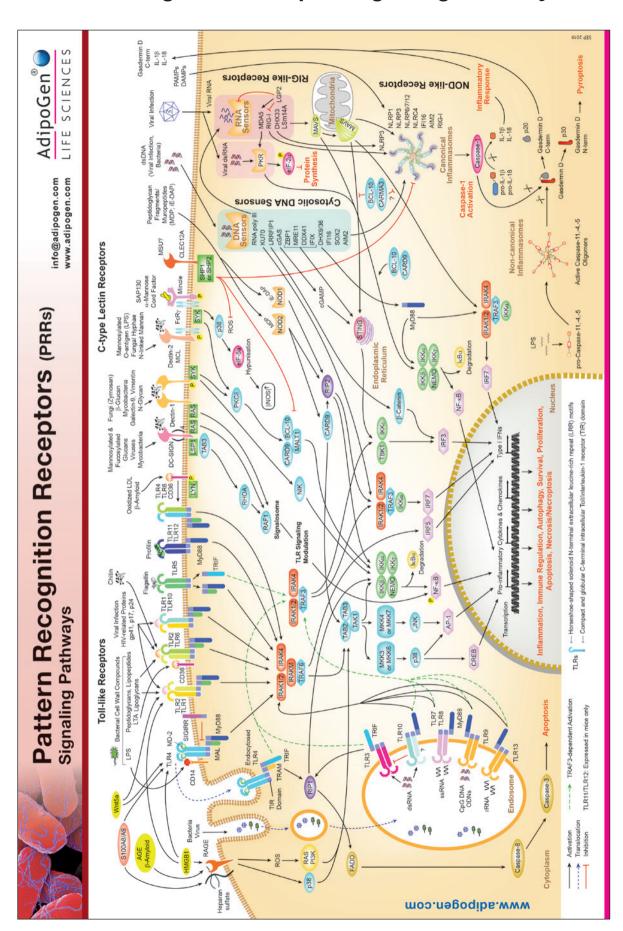
CONTENTS

Toll-like Receptor [TLR] Agonists
TLR1/TLR2 – Pam ₃ CSK ₄ 3
TLR3/MDA5 – Poly(I:C) 3
TLR4/CD14 – LPS 4–5
TLR5/TLR11/NLRC4 – Flagellin 3
TLR6/TL2 – FSL-1 3
TLR7/TLR8 – Nucleoside Analogs 3
TLR9 – ODNs 6–7
TLR11/12 – Profilin 3
RIG-I-like Receptors & Cytosolic DNA Sensor Agents
Poly(I:C), cGAMP 3
TLR4 and TLR9 Antagonists
IAXO Compounds, iODNs 4, 7
TLR Signaling Antibodies 8
Non-canonical Inflammasome 8 Caspase-11, Gasdermin
Pattern Recognition Receptor

Nod-like Receptors [NLRs] > See Inflammasome Brochure!

Signaling Pathways Chart

Pattern Recognition Receptor Signaling Pathways



Selected TLR Agonists & STING Ligand

TLR1/2 Complex Agonist

Pam₃Cys-Ser-(Lys)₄.3HCl

AG-CP3-0003

TLR3/MDA5 Agonist

Poly(I:C) (Endotoxin-free) (sterile)

IAX-200-021 2 mg | 5 mg | 3 x 5 mg | 5 x 5 mg

TLR11/TLR12 Agonist

Profilin (Toxoplasma gondii) (rec.)

AG-40B-0121 10 μg | 3 x 10 μg

STING Activator

cGAMP.2Na

AG-CR1-3588 100 μg | 500 μg

FSL-1 - The MALP-2 Alternative

TLR6/TLR2 Agonists

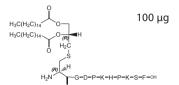
(R)-FSL-1

AG-CP3-0010

Formula: $C_{84}H_{140}N_{14}O_{18}S$

MW: 1666.2

CAS: 322455-70-9 (R/S)



Potent stimulator of TLR2/TLR6. The naturally occurring R-stereoisomer is biologically more active than the S-stereoisomer.

FSL-1

AG-CP3-0009 100 μg



TLR5/TLR11/NLRC4 Agonist - Flagellin & Mutants

2 mg

Toll-like receptor 5 (TLR5) recognizes **flagellin**, the principal component of bacterial flagella, from both Gram-positive and Gram-negative bacteria. Activation of the receptor stimulates the production of proinflammatory cytokines, such as TNF- α , through signaling via the adapter proteins MyD88, TIRAP and TRIF. Flagellin activates the innate immune system not only through the TLR5, but also through the intracellular NAIP5/NLRC4 (IPAF) inflammasome protein. In mice, Flagellin is also recognized by the cathepsin-cleaved TLR11, which is evolutionarily the closest member of the TLR5 gene family.

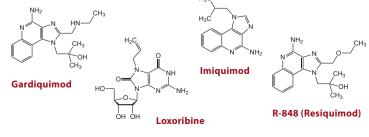
AdipoGen Life Sciences offers different types of **low endotoxin** and **high purity flagellins**, including **pathway specific mutants**. The Flagellin (NLRC4 Mutant) (rec.) (Prod. No. AG-40B-0126) is only detected by TLR5 but not by NLRC4, whereas the Flagellin (TLR5 Mutant) (rec.) (Prod. No. AG-40B-0127) is only detected by NLRC4.

PRODUCT NAME	PID	SIZE
Flagellin	AG-40B-0095	100 μg
Flagellin (high purity)	AG-40B-0025	10 µg 3 х 10 µg
Flagellin (rec.)	AG-40B-0125	10 µg 3 х 10 µg
NEW Flagellin (NLRC4 Mutant) (rec.)	AG-40B-0126	10 µg 3 х 10 µg
NEW Flagellin (TLR5 Mutant) (rec.)	AG-40B-0127	10 μg 3 x 10 μg

TLR7/TLR8 Agonists

Toll-like receptor 7 (TLR7) and TLR8 play an important role in the immune response to viral infection. They recognize single stranded RNAs as their natural ligand and also small synthetic molecules such as imidazoquinolines* and nucleoside** analogs. The TLR7 agonist Imiquimod can also contribute to NLRP3 activation through the inhibition of mitochondria complex I and through endolysosomal effects.

PRODUCT NAME	PID	SIZE	
TLR7 Agonists			
Gardiquimod *	AG-CR1-3583	5 mg 25 mg	
Imiquimod *	AG-CR1-3569	100 mg 250 mg	
Loxoribine **	AG-CR1-3584	5 mg 25 mg	
TLR7/8 Agonist & NLRP3 Activator			
R-848 (Resiguimod)*	AG-CR1-3582	5 mg 25 mg	



TLR4 Agonists - LPS, Lipid A and MPLA

Bacterial lipopolysaccharide (LPS) is the major structural component of the outer wall of all Gram-negative bacteria and a potent activator of the immune system. Activation of cells by LPS is mediated by the extracellular receptor Toll-like receptor 4 (TLR4) or by intracellular recognition and activation of caspase-11 (caspase-4/5 in humans) depending on extracellular or intracellular triggers, respectively. For optimal interaction with LPS, TLR4 requires association with myeloid differentiation protein 2 (MD-2). According to current consensus activation of TLR4 is preceded by the transfer of LPS to membrane-bound (m) or soluble (s) CD14 by LPS-binding protein (LBP). R-form LPS and lipid A, but not S-form LPS, are capable of inducing TNF- α responses also in the absence of CD14. LPS, synthesized by most wild-type (WT) Gram-negative bacteria (S-form LPS), consists of three regions, the O-polysaccharide chain, which is made up of repeating oligosaccharide units, the core oligosaccharide and the lipid A, which harbors the endotoxic activity of the entire molecule (see Figure 2). R-form LPS synthesized by the so-called rough (R) mutants of Gram-negative bacteria lacks the O-specific chain. The core oligosaccharide may be present in different degrees of completion, depending on the class (Ra to Re) to which the mutant belongs. LPS are amphipathic molecules whose hydrophobicity decreases with increasing length of the sugar part. Based upon these differences, S- and R-form LPS show marked differences in the kinetics of their blood clearance and cellular uptake as well as in the ability to induce oxidative burst in human granulocytes and to activate the host complement system. S-form LPS is the prefered in vivo TLR4 agonist, whereas R-form LPS can activate TLR4 on a variety of cells in vitro, which do not express CD14 or where the cell culture medium does not contain sufficient quantities of soluble CD14 or LBP.

LIT: CD14 is required for MyD88-independent LPS signaling: Z. Jiang, et al.; Nat. Immunol. **6**, 565 (2005) • R-form LPS, the master key to the activation of TLR4/MD-2-positive cells: M. Huber, et al.; Eur. J. Immunol. **36**, 701 (2006)

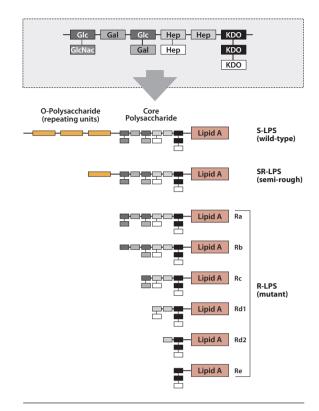


FIGURE 2: Schematic representation of the different LPS chemotypes: GlcNAc = N-Acetyl-glucosamine; Glc = Glucose; Gal = Galactose; Hep = Heptose; KDO = 2-Keto-3-desoxyocto-nate. Adapted from M. Huber, et al.; Eur. J. Immunol. **36**, 701 (2006).

TLRpure™ LPS from Innaxon – The TLR4/LPS Experts – Highest Quality

- TLRpure™: Qualified Purity & Activity
- High potency TLR4-specific ligands
- Ultrapure (no detectable protein, RNA & DNA)
- Standardized aqueous sterile solutions
- Tested on TLR4 KO murine macrophages
- . BULK available for in vivo studies
- Excellent lot-to-lot reproducibility
- Broadest LPS chemotype and serotype selection



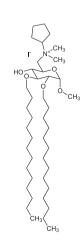


IAXO Compounds – Inhibitors of Sterile Inflammation

The novel IAXO compounds are synthetic TLR4/CD14 ligands with TLR4 modulating activities *in vitro* and conferring protection against TLR4/CD14-mediated tissue damage and inflammation *in vivo* [1-3]. IAXOs are useful to explore CD14-dependent and TLR4-independent pathways and TLR4 activation by endogenous ligands (e.g. hyaluronic acid oligosaccharides, oxLDL, HMGB1) in sterile inflammation. IAXO compounds have been shown to inhibit neuropathic pain [1], secondary necrosis of acute drug-induced liver failure [2] and vascular inflammation and abdominal aortic aneurysm [3] by blocking non-hematopoietic TLR4 signaling. They are useful tools, where inhibition of sterile (auto-) inflammation is desired, without compromising TLR4's key role in the defense of pathogens.

LIT: [1] I. Bettoni, et al.; Glia 56, 1312 (2008) • [2] N. Shah, et al.; Gut 61, A28 (2012) • [3] C. Huggins, et al.; Atherosclerosis 241, e53 (2015)

PRODUCT NAME	PID	SIZE
IAXO-101 (CD14/TLR4 Antagonist) (synthetic)	IAX-600-001	1 mg 5 mg
IAXO-102 (CD14/TLR4 Antagonist) (synthetic)	IAX-600-002	1 mg 5 mg
IAXO-103 (CD14/TLR4 Antagonist) (synthetic)	IAX-600-003	1 mg 5 mg



TLR4 Agonists: Homogenous Ready-to-Use LPS Solutions



PRODUCT NAME	PID	SIZE
S-form LPS		
LPS from E. coli O8:K27 (S-form) TLR <i>pure</i> ™ Sterile Solution	IAX-100-006	500 μg 1 mg 5 x 1 mg
LPS from E. coli O111:B4 (S-form) TLRpure™ Sterile Solution	IAX-100-012	500 μg 1 mg 5 x 1 mg
LPS from E. coli O55:B5 (S-form) TLR <i>pure</i> ™ Sterile Solution	IAX-100-013	500 μg 1 mg 5 x 1 mg
THE STANDARD LPS (Universal)* from S. abortus equi (S-form) TLRpure™ Sterile Solution	IAX-100-009	500 μg 1 mg 5 x 1 mg
LPS from S. enteritidis (S-form) TLR <i>pure</i> ™ Sterile Solution	IAX-100-019	500 μg 1 mg
LPS from S. minnesota (S-form) TLR <i>pure</i> ™ Sterile Solution	IAX-100-020	500 μg 1 mg 5 x 1 mg
LPS from S. typhimurium (S-form) TLR <i>pure™</i> Sterile Solution	IAX-100-011	500 μg 1 mg 5 x 1 mg
R-form LPS		
LPS from E. coli EH100 (Ra) TLR <i>pure</i> ™ Sterile Solution	IAX-100-010	500 μg 1 mg 5 x 1 mg
LPS from E. coli J5 (Rc) TLR <i>pure</i> ™ Sterile Solution	IAX-100-014	500 μg 1 mg 5 x 1 mg
THE STANDARD LPS from E. coli R515 (Re) TLRpure™ Sterile Solution	IAX-100-007	500 μg 1 mg 5 x 1 mg
THE STANDARD LPS from S. minnesota R595 (Re) TLRpure™ Sterile Solution	IAX-100-008	500 μg 1 mg 5 x 1 mg
LPS from S. minnesota R345 (Rb) TLR <i>pure</i> ™ Sterile Solution	IAX-100-015	500 μg 1 mg 5 x 1 mg
LPS from S. minnesota R60 (Ra) TLR <i>pure</i> ™ Sterile Solution	IAX-100-016	500 μg 1 mg
LPS from S. minnesota R5 (Rc) TLR <i>pure</i> ™ Sterile Solution	IAX-100-017	500 μg 1 mg 5 x 1 mg
LPS from S. minnesota R7 (Rd1) TLR <i>pure</i> ™ Sterile Solution	IAX-100-018	500 μg 1 mg 5 x 1 mg
LPS from S. minnesota R3 (Rd2) TLR <i>pure</i> ™ Sterile Solution	IAX-100-021	500 μg 1 mg 5 x 1 mg
Lipid A und MPLA		
Lipid A from E. coli R515 (Re) TLR <i>pure</i> ™ Sterile Solution	IAX-100-004	250 μg 500 μg 1 mg
Lipid A from S. minnesota R595 (Re) TLR <i>pure</i> ™ Sterile Solution	IAX-100-001	250 μg 500 μg 1 mg
MPLA from E. coli R515 (Re) TLR <i>pure</i> ™ Sterile Solution	IAX-100-003	250 μg 500 μg 1 mg
MPLA from S. minnesota R595 (Re) TLR <i>pure</i> ™ Sterile Solution	IAX-100-002	250 μg 500 μg 1 mg

 $^{^*}$ Universal: High-stability LPS solution with long-term consistent high TLR4-exclusive potency in immune activation.

Synthetic TLR4 Agonists - Kdo2-Lipid A and MPLA

PRODUCT NAME	PID	SIZE
Kdo2-Lipid A (ready-to-use)	AG-CU1-0001	1 mg
MPLA (synthetic) Sterile Solution	AG-CU1-0002	100 μg

TLR4 Signaling Pathway Regulatory Proteins

High mobility group box 1 (HMGB1) is a Damage-Associated Molecular Pattern (DAMP) that interacts with multiple pattern recognition receptors (PRRs). Many of its effects in sterile inflammation models occur through an interaction with the TLR4/myeloid differentiation protein 2 (MD-2) complex. SIGIRR negatively regulates TLR4 signaling pathways by interacting transiently with TLR4, IRAK4 and TRAF6. The ST2 receptor, a member of the Toll/IL-1 receptor superfamily, has been shown to function as a negative regulator of TLR4 and TLR2 signaling by sequestering MAL and MyD88. CD14 acts as a co-receptor with TLR4/MD-2 for the detection of bacterial lipopolysaccharide. CD36 is a scavenger receptor shown to promote sterile inflammation through assembly of a TLR4/ TLR6 heterodimer upon binding to oxLDL or β -Amyloid.

PROTEINS	PID
HMGB1 (human) (rec.) (His)	CHI-HR-200HMGB1
HMGB1 (rat) (rec.) (His)	CHI-RR-300HMGB1
HMGB1 (rat):Fc (human) (rec.)	CHI-RF-311HMGB1
SIGIRR (human):Fc (human) (rec.)	AG-40A-0093T
ST2 (human):Fc (human) (rec.)	AG-40A-0059
IL-33R (human):Fc (human) (rec.)	CHI-HF-21033R
IL-33R (mouse):Fc (mouse) (rec.)	CHI-MF-11033R

PROTEINS	PID
anti-CD14 (human), mAb (UCHM1)	ANC-163-020
anti-CD36 (human), mAb (SMO)	ANC-185-020
anti-ST2 (human), pAb	AG-25A-0058
anti-ST2 (human), pAb (ATTO 488)	AG-25A-0058YTD
anti-ST2 (human), pAb (ATTO 647N)	AG-25A-0058YTS
anti-ST2 (human), mAb (ST33868)	AG-20A-0044
anti-HMGB1, mAb (rec.) (Giby-1-4)	AG-27B-0002

TLR9 Agonists – Unmethylated CpG Dinucleotides [ODNs]

Unmethylated CpG dinucleotides within particular sequence contexts are responsible for the immunostimulatory activity of bacterial DNA. Synthetic oligonucleotides (ODN), that contain such CpG motifs (CpG ODNs), mimic microbial DNA and are detected by Toll-like receptor 9 (TLR9).

Endotoxin-free & Sterile ODNs from Innaxon

- TLRpure™: Qualified Purity & Activity
- Endotoxin-free and sterile
- Potent TLR9 ligands

- Tested on TLR9 KO murine macrophages
- BULK available for in vivo studies (pre-clinical grade)
- ddWater Endotoxin-free (sterile) included



Stimulatory ODNs (CpG ODNs)

Different types of CpG ODNs were identified based on their biological effects on different cell types through TLR9: **ODN Type A** is a potent inducer of IFN- α in human PDC, leading to antigen presenting cell (APC) maturation, whereas **ODN Type B** is a weak inducer of IFN- α but rather stimulates IL-8 production and increasing costimulatory and Ag-presenting molecules and triggers proliferation of B cells and IL-6 production. A third type of CpG ODN, termed **ODN Type C**, shows high induction of INF- α in PDC and activation of B cells.

PRODUCT NAME	PID	SIZE
ODN 1668 (Type B) Endotoxin-free (sterile)	IAX-200-001	100 μg 1 mg 3 x 1 mg
THE STANDARD ODN 1826 (Type B) Endotoxin-free (sterile)	IAX-200-002	100 μg 1 mg 3 x 1 mg
THE STANDARD ODN 2216 (Type A) Endotoxin-free (sterile)	IAX-200-005	100 μg 1 mg 3 x 1 mg
ODN 1585 (Type A) Endotoxin-free (sterile)	IAX-200-003	100 μg 1 mg 3 x 1 mg
ODN M362 (Type C) Endotoxin-free (sterile)	IAX-200-004	100 μg 1 mg
ODN 2006 (Type B) Endotoxin-free (sterile)	IAX-200-006	100 μg 1 mg 3 x 1 mg
ODN 2395 (Type C) Endotoxin-free (sterile)	IAX-200-007	100 μg 1 mg 3 x 1 mg

Control ODNs (GpC ODNs)

Inactive control compounds for CpG ODNs do not stimulate TLR9. They are composed of the same sequence as their stimulatory counterparts, but instead of CpG they contain GpC dinucleotides.

PRODUCT NAME	PID	SIZE
ODN 1720 (Control for ODN 1668) Endotoxin-free (sterile)	IAX-200-200	100 μg
ODN 2138 (Control for ODN 1826) Endotoxin-free (sterile)	IAX-200-201	100 μg
Neutral-ODN (Control for iODNs) Endotoxin-free (sterile)	IAX-200-202	100 μg 1 mg 3 x 1 mg
ODN 2118 (Control for ODN 1585) Endotoxin-free (sterile)	IAX-200-203	100 μg
ODN M383 (Control for ODN M362) Endotoxin-free (sterile)	IAX-200-204	100 μg
ODN 2243 (Control for ODN 2216) Endotoxin-free (sterile)	IAX-200-205	100 μg
ODN 2137 (Control for ODN 2006) Endotoxin-free (sterile)	IAX-200-206	100 μg



TLR9 Antagonists — Inhibitory ODNs (iODNs)

In recent years several groups have studied the sequence requirements, specificity, signaling pathways and kinetics of the Toll-like receptor 9 (TLR9) suppression by inhibitory oligonucleotide motifs, which led to a class of novel inhibitory oligonucleotides (iODNs). Subsequently it has been discovered that telomeric DNA repeats (TTAGGG)_n can block immune activation by CpG-ODNs. Consequently, a classification for iODNs has been proposed.

- Class I: G-stretch ODNs: TLR9-specific competitors, some iODNs may also affect TLR7 and TLR8 signaling.
- Class II: ODNs with telomeric repeats: TLR-independent inhibitors of STAT signaling (cellular uptake via an "ODN receptor"?)
- Class III: Inhibitors of DNA uptake in a sequence independent manner.
- Class IV: Long phosphorothioate ODNs as direct competitors of TLR9 signaling in a sequence independent manner.

LIT: Immunotherapeutic utility of stimulatory and suppressive oligodeoxynucleotides: K.J. Ishii, et al.; Curr. Opin. Mol. Ther. **6,** 166 (2004) • Inhibitory oligodeoxynucleotides - therapeutic promise for systemic autoimmune diseases?: P. Lenert; Clin. Exp. Immunol. **140,** 1 (2005) • DNA motifs suppressing TLR9 responses: A. Trieu, et al.; Crit. Rev. Immunol. **26,** 527 (2006)

iODNs - Potent Inhibitors of TLR9 Signaling

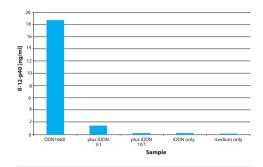


FIGURE 3: ODN 1668 (Prod. No. IAX-200-001) was added at 50pM/ml to macrophages in a 96-well plate simultaneously together with iODN 2088 (Prod. No. IAX-200-050) at the indicated molar excess, cell supernatants harvested after 24 hours and IL-12-p40 analyzed by cytokine ELISA.

PRODUCT NAME	PID	SIZE
iODN (inhibitory ODN) 2088 Endotoxin-free (sterile)	IAX-200-050	100 μg 1 mg 3 x 1 mg
iODN (inhibitory ODN) (ttaggg) ₄ Endotoxin-free (sterile)	IAX-200-051	100 μg 1 mg 3 x 1 mg
G-type iODN (inhibitory ODN) Endotoxin-free (sterile)	IAX-200-052	100 μg 1 mg
Mini-iODN (inhibitory ODN) Endotoxin-free (sterile)	IAX-200-053	100 μg 1 mg
Mega-iODN (inhibitory ODN) Endotoxin-free (sterile)	IAX-200-054	100 μg 1 mg
Duo-iODN (inhibitory ODN) Endotoxin-free (sterile)	IAX-200-055	100 μg 1 mg

[▶] All ODNs include 1 vial of ddWater Endotoxin-free (sterile) (IAX-900-002).

TECHNICAL NOTE

Contaminations in TLR Ligands

Specific recognition of different PAMPs by TLRs revealed that only the purest ligands, free of any other immuno-stimulatory contamination, allow to successfully elucidate the role of each TLR. While LPS was thought to not only activate TLR4 but also TLR2, repurification of commercial preparations of both *E. coli* and *Salmonella minnesota* LPS showed that this LPS no longer induces cellular activation through TLR2 [1]. Furthermore it has been shown that purified peptidoglycans activate Nod1 and do not involve TLR2 or TLR4 [2]. Synthetic CpG ODNs (contaminated with LPS) show different activation of certain immune cell subsets.

LIT : [1] Repurification of lipopolysaccharide eliminates signalling through both human and murine toll-like receptor 2: M. Hirschfeld, et al.; J. Immunol. **165**, 618 (2000) • **[2]** Toll-like receptor 2-dependent bacterial sensing does not occur via peptidoglycan recognition: L. H. Travassos, et al.; EMBO Rep. **5**, 1000 (2004)



offers endotoxinfree and sterile buffers to avoid contaminations upon solubilization of powder form ligands!

PRODUCT NAME	PID	SIZE
PBS Endotoxin-free (sterile)	IAX-900-001	1.5 ml 10 ml 3 x 10 ml 100 ml
ddWater Endotoxin-free (sterile)	IAX-900-002	1.5 ml 10 ml 3 x 10 ml 100 ml
Physiological Saline [Sodium Chloride 0.9%] Sterile Solution	IAX-900-003	1.5 ml 10 ml 3 x 10 ml 100 ml

TLR Signaling Antibodies

See www.adipogen.com for Labeled Antibodies!

ANTIBODIES	PID	SIZE	APPLICATIONS	SPECIES
anti-TLR2, mAb (ABM3A87)	AG-20T-0300	100 μg	FACS, IHC, WB	Hu, Ms
anti-TLR3 (human), mAb (ABM15D5)	AG-20T-0301	100 μg	FACS, WB	Hu
anti-TLR3 (mouse), mAb (ABM24E5)	AG-20T-0302	100 μg	FACS, WB	Ms
anti-TLR4 (human), mAb (ABM19C4)	AG-20T-0303	100 μg	FACS, IHC, WB	Hu
anti-TLR5 (human), mAb (ABM22G1)	AG-20T-0304	100 µg	FACS, IHC	Hu
anti-TLR6 (human), mAb (ABM1B50)	AG-20T-0305	100 μg	FACS, WB	Hu
anti-TLR7 (human), mAb (ABM2C27)	AG-20T-0306	100 μg	FACS, IHC, WB	Hu
anti-TLR8 (human), mAb (ABM15F6)	AG-20T-0307	100 μg	FACS, IHC	Hu
anti-TLR9, mAb (ABM1C51)	AG-20T-0308	100 μg	FACS, IHC, WB	Hu, Ms
anti-TLR9 (mouse), mAb (ABM4D70)	AG-20T-0309	100 μg	FACS, WB	Ms
anti-TLR10 (human), mAb (ABM3C85)	AG-20T-0310	100 µg	WB	Hu

C-type Lectin Receptors - Dectin-1 & -2

Dectin-1 and Dectin-2 are type II transmembrane C-type lectin pattern recognition receptors that recognize β -glucans and other fungal structures through the carbohydrate-recognition domain (CRD). They function by binding to cell walls of different fungal species triggering intracellular responses like phagocytosis and cytokine production.

PROTEINS	PID
Fc (human):Dectin-1 (mouse) (rec.)	AG-40B-0138
Fc (human):Dectin-2 (mouse) (rec.)	AG-40B-0139

Non-canonical Inflammasome

The inflammasome is a large cytoplasmic multiprotein complex scaffolded by a pathogen recognition receptor (PRR) that senses infections and endogenous dangers. The inflammasome signals activation of caspase-1, and active caspase-1 then processes IL-1 β /IL-18 and triggers a lytic form of programmed necrosis called pyroptosis to mount innate immune responses. In addition Gram-negative bacteria can trigger a caspase-11-dependent (in human caspase-4/5) pathway to induce pyroptosis as well, called the non-canonical inflammasome.

PRODUCT NAME	PID	SIZE	APPLICATIONS	SPECIES
anti-Caspase-4 /11 (p20), mAb (Flamy-1)	AG-20B-0060	100 μg	IP, WB	Hu, Ms
anti-Caspase-11 (mouse), mAb (4E11)	AG-20T-0140	100 µg	FACS, IP, WB	Ms
anti-Caspase-11 (mouse), mAb (8A5)	AG-20T-0139	100 µg	FACS, IP, WB	Ms
anti-Gasdermin D (mouse), pAb (IN110)	AG-25B-0036	100 µg	WB	Ms
Gasdermin D (mouse) ELISA Kit	AG-45B-0011	96 wells	ELISA	Ms

Gasdermin D Inhibitors

PRODUCT NAME	PID	SIZE
Necrosulfonamide	AG-CR1-3705	5 mg 25 mg
U-73122	AG-CR1-3698	1 mg 5 mg



See our Inflammasome Brochure!



TEL +41-61-926-60-40 FAX +41-61-926-60-49 info@adipogen.com

NORTH & SOUTH AMERICA Adipogen Corp.

TEL +1-858-457-8383 FAX +1-858-457-8484 info-us@adipogen.com