InvivoGen insight

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NLRP1 & NLRP3: a tale of two inflammasome sensors

Early 2000s, NLRP1 and NLRP3 were the first described inflammasomes, consisting in cytosolic multi-protein complexes that sense danger signals. Activated inflammasomes operate the maturation and release of the pro-inflammatory cytokines IL-1β and IL-18[1]. Although NLRP1 was the first identified member of the NLR (Nucleotide-binding domain and Leucine-rich repeat Receptor) family, its functional relevance remained obscure for many years. Meanwhile, NLRP3 rapidly became the prototypical and best characterized inflammasome, being linked to a long list of diseases and conditions, including type-2 diabetes, gouty arthritis, and cancer[2]. Today, NLRP1 research is reaching a new momentum. Viral infections[3,4] and UVB radiation[5] have been identified as natural NLRP1 triggers in humans. Moreover, excessive NLRP1 activation contributes to COVID-19 severity in the context of SARS-CoV-2 infection[4], and NLRP1 gain-of-function mutations result in increased squamous cell carcinoma susceptibility[6]. It is tempting to speculate that these new findings might dethrone NLRP3 in the inflammasome realm. Indeed, in some diseases, NLRP3 activation may be a bystander outcome from other inflammasome responses. Deciphering the roles of NLRP1 and NLRP3 in homeostatic and pathological contexts will lead to new therapeutics for microbial and non-infectious diseases. Here, we review the current knowledge about these two sensors and discuss the experimental difficulty in assessing their respective role in patho-physiological contexts.

Of mice and men

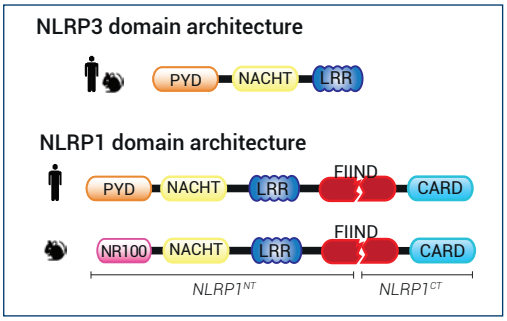
NLRP3 and NLRP1 differ by many aspects, including tissue expression, protein structure, and activation mechanisms. Moreover, while NLRP3 is highly conserved between humans and mice, it is not the case for NLRP1.

• Expression

In humans, a single *NLRP1* gene is alternatively spliced, isoform 1 being the best studied[7]. In contrast, mice exhibit a multi-gene configuration. Most of our current knowledge has focused on the murine *Nlrp1b* paralog[8]. Human NLRP1 is highly expressed in epithelial barriers (i.e. keratinocytes and bronchial epithelial cells), whereas mouse NLRP1B and human/mouse NLRP3 are mainly found in myeloid cells[8].

• Architecture

NLR proteins typically feature an N-terminal effector domain, a central nucleotide-binding domain (NACHT) and a C-terminal ligand-binding region that is composed of several leucine-rich repeats (LRRs). The NLRP and NLRC subgroups differ in their N-termini with a pyrin domain (PYD) and a caspase recruitment domain (CARD), respectively.



NLRP3 comprises a PYD-NACHT-LRR module, where the NACHT and LRR domains interact to maintain NLRP3 in an auto-repressed/closed conformation. PYD drives the inflammasome formation upon NLRP3 activation[9].

NLRP1 architecture contrasts with NLRP3 and other NLRP proteins. In addition to a PYD-NACHT-LRR module in N-terminal , it features a function-to-find-domain (FIIND) upstream of a CARD domain in C-terminal. FIIND undergoes constitutive auto-proteolysis, generating a "licensed" full-length NLRP1 ($NLRP1^{FL}$), in which the N-terminal ($NLRP1^{NT}$) and C-terminal ($NLRP1^{CT}$) fragments remain non-covalently associated[10]. Structural studies have revealed a ternary complex where one $NLRP1^{FL}$ and one $NLRP1^{CT}$ bind to dipeptidyl peptidases, DPP8/9, keeping the inflammasome-forming $NLRP1^{CT}$ in check[11]. How the single $NLRP1^{CT}$ is initially generated is unknown. Upon $NLRP1^{CT}$ release, CARD drives NLRP1 inflammasome formation. Of note, in mice, PYD is replaced by a NR100 domain.

> It is tempting to speculate that these new findings might dethrone NLRP3 in the inflammasome realm.

• NLRP3 inflammasome formation

NLRP3 can sense a wide range of stimuli (microbial, sterile, or environmental). Yet, no NLRP3 ligand *per se* has been described and the exact mechanisms underlying NLRP3 activation remain to be fully elucidated. As of today, the paradigm is that cytosolic stress signals such as K^+ efflux and oxidized mitochondrial DNA contribute to NLRP3 post-translational modifications and opening[12,13]. This change in NLRP3 conformation allows PYD to recruit pro-caspase-1 via ASC, a PYD/CARD-containing adaptor[12]. The NimA-related protein kinase 7 (NEK7) interacts with LRR and has been proposed to support NLRP3 oligomerization in mice[14,15], while it is dispensable in humans[16]. Importantly, NLRP3 activation steps seem highly conserved in humans and mice, allowing the study of NLRP3 relevance using murine disease models.

> Since 2020, our understanding of human NLRP1 activation has accelerated. Most reports are in line with a "functional degradation" model [...]

• NLRP1 inflammasome formation

A major gap in the comprehension of NLRP1 inflammasome was filled in 2012 when the anthrax lethal factor (LF) was shown to activate NLRP1 and drive pro-inflammatory responses upon *B. anthracis* infection in a mouse model[17]. However, this finding did not recapitulate in humans[10]. Since 2020, our understanding of human NLRP1 activation has accelerated. Most reports are in line with a "functional degradation" model: distinct stimuli, listed below, trigger destabilization and proteasomal degradation of the NLRP1^{NT} portion, allowing the release of the NLRP1^{CT} fragment.

– ***Val-boroPro (VbP, Talabostat)***, a small molecule known to stimulate anti-cancer immune responses, inhibits DPP8/9's catalytic activity and induces NLRP1^{NT} degradation both in mice and humans[11,18]. Of note, there is currently no (patho) physiogical-encoded molecule identified to act as a DPP8/9 inhibitor.

– ***Pathogen-encoded proteins***, notably proteases such as 3Cpro from the human rhinovirus, the causative agent of common cold[3], and NSP5 from SARS-CoV-2, the causative agent of COVID-19[4], have been shown to induce NLRP1^{NT} degradation and NLRP1 activation. Hence, NLRP1 has been suggested to act as a broad decoy receptor of microbial virulence factors. These triggers are restricted either to human or mouse NLRP1, illustrating host and pathogen co-evolution.

– ***Ultraviolet B (UVB) radiation*** causes a ribotoxic stress response mediated by the MAP3 kinase ZAKα. This kinase initiates a hyperphosphorylation in NLRP1^{NT}, which possibly accelerates its degradation[5].

– ***Long double-stranded (ds)RNA***, generated upon replication of the Semiliki Forest Virus triggers human, but not mouse, NLRP1 activation[19]. In this case, NLRP1 may switch conformation, allowing NLRP1^{NT} degradation[19].

More research is still needed to identify the mechanisms by which the NLRP1^{CT} fragment is released.

• NLRP3 and NLRP1 safeguards

Sensor activation and inflammasome formation lead to rapid and powerful inflammatory responses, extremely harmful if accidentally triggered. Thus, to prevent unwanted activation, multiple safeguards exist at all steps of the inflammasome response. Such checkpoints have been described at the sensor level, at least for NLRP3 and NLRP1.

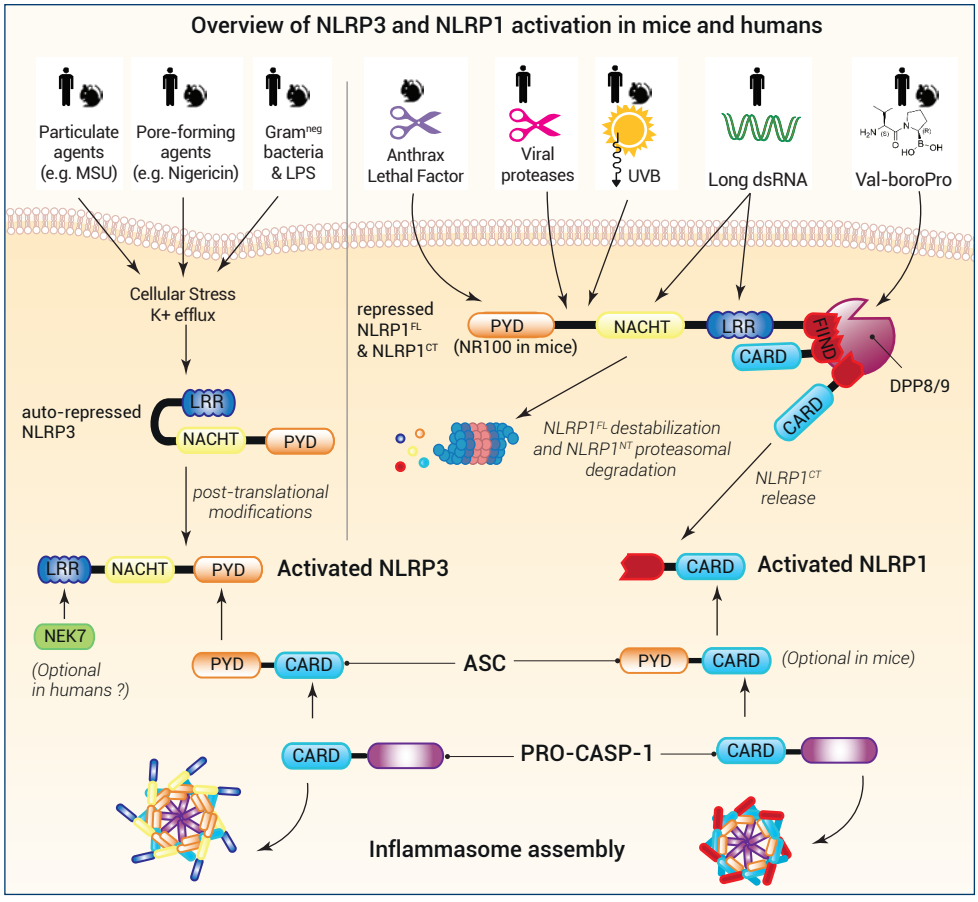
NLRP3 activation is governed by two signals. A first priming signal renders the NLRP3 sensor responsive to a second activatory signal. The priming signal can be delivered by PRRs (pathogen recognition receptors) or cytokine receptors. Depending on the cell type, as well as the signal nature and duration, NLRP3 priming occurs through transcriptional upregulation, post-translational modifications, or binding with a protein partner, such as NEK7[13,20]. Recently, Schmacke *et al.* have identified an IKKβ-mediated NEK7-independent and predominant priming pathway for NLRP3 in humans[16]. The authors show that IKKβ induces NLRP3 priming by increasing its recruitment to the trans-golgi network (TGN), an organelle thought to play a key role in NLRP3 inflammasome assembly[12,16,21].

NLRP1 activation is governed by the release of the functional NLRP1^{CT} fragment from the ternary complex DPP8/9-NLRP1^{FL}-NLRP1^{CT}, which has been suggested as a checkpoint for NLRP1 inflammasome formation[11,22]. A recent report by Ball *et al.* indicates that during homeostasis, NLRP1^{FL} is associated with oxidized thioredoxin-1 (TRX1), and that reductive stress triggers their dissociation and the proteasomal degradation of the NLRP1^{NT} portion. The resulting NLRP1^{CT} fragment is sequestered and kept in check within the ternary complex until a second signal triggers its release for inflammasome assembly[22]. These *in vitro* data strongly suggest that a reductive stress potentiates NLRP1 activation. Yet, additional research is needed to identify physiological inducers of reductive stress. Moreover, it is unclear whether the NLRP1^{NT} degradation upon reductive stress acts as a priming step, or if it induces the NLRP1 inflammasome formation. In both cases, it may be the accumulation of NLRP1^{CT} after degradation of NLRP1^{NT} by reductive stress and other stimuli (e.g. viral proteases, UVB radiation) that unlocks the ternary complex checkpoint[11,22].

The frontier between the priming and activation for NLRP3 or NLRP1 sensors upstream the inflammasome formation remains a major research axis.

• Signaling

Activated NLRP3 and NLRP1 sensors associate with ASC through their PYD and CARD domain, respectively, to recruit pro-caspase-1[12,23]. In humans, pro-caspase-1 interaction with NLRP1 is strictly ASC-dependent, while it is dispensable in mice[23]. Ultimately, this complex causes caspase-1 activation, which in turn, cleaves pro-IL-1β



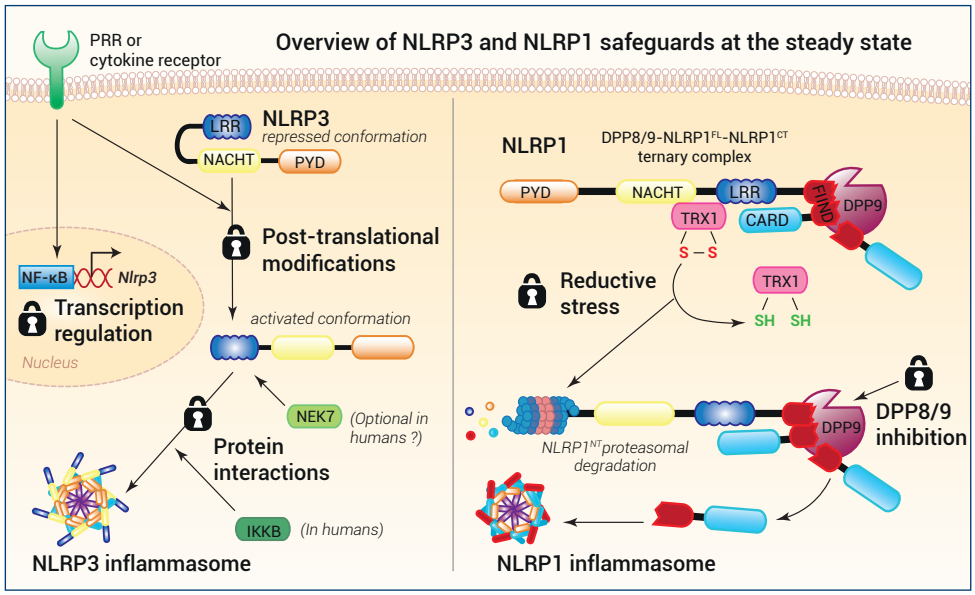
and pro-IL-18, as well as Gasdermin D (GSDMD) into their biologically active forms. The formation of GSDMD pores at the plasma membrane allows the secretion of IL-1β and IL-18, and eventually leads to pyroptotic cell death[24].

Of note, NLRP3 and NLRP1 inflammasomes may use additional signaling mechanisms. In mice harboring an inactive/knockout caspase-1 or GSDMD, the NLRP3 inflammasome engages a delayed, alternative cell death, mediated by caspase-8, caspase-3, and Gasdermin E (GSDME)[25,26]. In human lung epithelial cells, the SARS-CoV-2 protease NSP5 counteracts NLRP1 inflammasome signaling by inactivating GSDMD. The infected cell death is ensured by NLRP1-driven activation of caspase-3 and GSDME cleavage[4]. Interestingly, patients with severe COVID-19 display elevated plasmatic levels of inflammasome markers (IL-18, GSDMD, GSDME, caspase-3) and mature IL-16, a caspase-3-derived alarmin[4]. This finding warrants future *in vivo* studies to decipher the role and mechanisms of action for IL-16, for which little is known. Moreover, whether the caspase-3-GSDME axis is a compensatory or parallel signaling pathway for the cytokine release and/or the lytic phase, remains to be elucidated for each inflammasome[26].

Future challenges

Inflammasomes are beneficial in clearing pathogens, but their excessive stimulation can be detrimental to the host[2,27,28]. Thus, it is crucial to develop fine therapeutic strategies, avoiding systemic blockade of inflammatory cytokines and the resulting opportunistic infections. Severe COVID-19 may illustrate exacerbated NLRP3 responses. Reports show NLRP3 co-staining with inflammasome specks in patient monocytes[29]. NLRP3 implication in the lung pathology has also been demonstrated using a mouse model of SARS-CoV-2 infection[30]. However, the recent finding of NLRP1 activation by the SARS-CoV-2 NSP5 protease is calling for cautious experimental design. Is there a possibility that the data implying a direct NLRP3 response to the virus may also be attributed to the cellular stress caused by the NLRP1 inflammasome response? Likewise, NLRP3 activation may be triggered by the non-canonical caspase-4/5 response to LPS from bacterial secondary infection, a common event in respiratory viral infections[31].

Deciphering the relative importance of each inflammasome and feedfoward loops during the course of infections and pathogenicities requires *in vivo* studies. To circumvent the poor NLRP1 conservation between mice and humans, one strategy would be to use humanized mice where murine inflammasome elements are replaced by the transgenic expression of their human counterparts.



> The frontier between the priming and activation for NLRP3 or NLRP1 sensors upstream the inflammasome formation remains a major research axis.

In the absence of infections or sterile inflammation, gain-of-function (GOF) mutations in *NLRP3* and *NLRP1* cause monogenic human inflammatory diseases (i.e. inflammasomopathies)[32,33]. Hence, such mutations in *NLRP3* cause cryopyrin-associated periodic syndromes (CAPS), while GOF mutations in *NLRP1* cause conditions such as NLRP1-associated autoinflammation with arthritis (NAIAD) and multiple self-healing palmoplantar carcinoma (MSHPC)[32,33].

Interestingly, most of NLRP3 and NLRP1 autoinflammatory mutations lie in domains that are crucial for the sensor inhibition[32,33]. Therefore, they provide a first testing ground to screen small molecules for preventing inflammasome hyperactivation, by targeting specific sensors. Murine inflammatory disease models allowed the screening and discovery of MCC950, a potent and specific NLRP3 inhibitor[34,35]. This promising drug has been tested in phase II in clinical trials and is awaiting for further tests. Still, it has inspired pharmaceutical companies which now have advanced clinical trials of several other NLRP3 inhibitors[36].

Many questions remain opened and more studies are needed to relieve the burden of inflammasome linked diseases on public health.

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NLRP1 inflammasome cellular assays

InvivoGen offers a series of A549 human lung carcinoma-derived reporter cell lines to facilitate the research of the human NLRP1 inflammasome. The stable expression of NLRP1 along with an NF-κB-inducible ASC::GFP fusion protein allows the realtime monitoring of ASC speck formation using fluorescence microscopy. To help investigate the role of NLRP1 in SARS-CoV-2 infection, two cell lines stably overexpress the genes encoding for the SARS-CoV-2 receptors, human ACE2 and TMPRSS2.

NLRP1 reporter cells

- **A549-ASC-NLRP1 Cells** NEW
- **A549-ASCoV2-NLRP1 Cells** NEW
- **A549-ASC Cells (control)** NEW
- **A549-ASCoV2 Cells (control)** NEW

- Stable expression of human NLRP1 (except control cells)
- NF-κB-dependent expression of ASC::GFP
- ASCoV2 cells permissive to the SARS-CoV-2 virus

In **A549-ASC-NLRP1 cells**, induction of ASC::GFP expression using an NF-κB activator (e.g. hTNF-α) and NLRP1 activation using Val-boroPro (VbP, see below), trigger the formation of fluorescent ASC specks (visible as green dots) *(Fig. 1A)* and ultimately, pyroptotic cell death *(Fig. 1E)*.

A549-ASCoV2-NLRP1 cells, which also express the SARS-CoV-2 receptors, display similar responses upon viral infection *(Fig. 1B&F)*.

In **A549-ASC & A549-ASCoV2 control cells**, the lack of endogenous NLRP1 expression precludes the formation of ASC::GFP specks *(Fig. 1C&D)*, as well as pyroptosis *(Fig. 1E&F)*.

NLRP1 inflammasome inducer

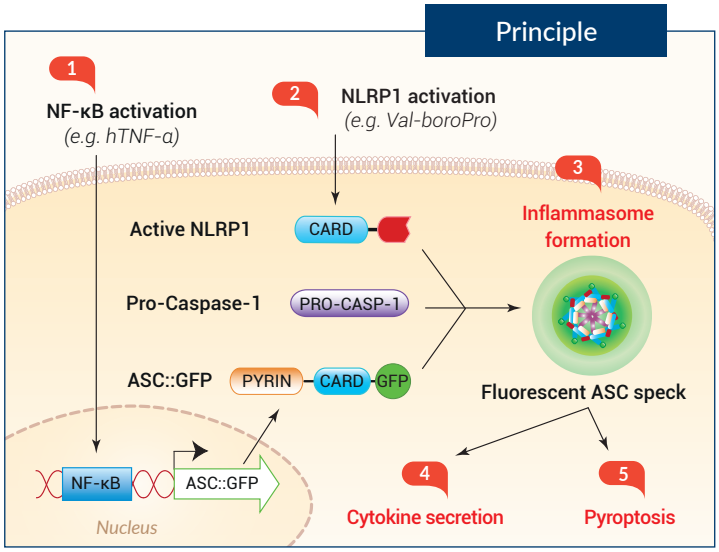
- **Val-boroPro** NEW

- Potent activator of NLRP1
- Highly pure and free of bacterial contamination
- Functionally tested

InvivoGen offers Val-boroPro (VbP), a non-selective dipeptidylpeptidase (DPP) inhibitor able to activate the NLRP1 inflammasome[1]. VbP triggers the NLRP1 inflammasome assembly, as visualized by ASC speck formation *(Fig. 1A)*, and lytic cell death *(Fig. 1E)*.

Bauernfried *et al.* recently showed that NLRP1 senses long dsRNA generated in the context of infection by the Semliki Forrest Virus[2]. This can be mimicked by transfection of **high molecular weight Poly(I:C)**. Data available on our website: www.invivogen.com/a549-ascg-nlrp1

PRODUCTS	QTY	CAT. CODE
A549-ASC-NLRP1 Cells	3-7 x 10^6 cells	a549-ascg-nlrp1
A549-ASCoV2-NLRP1 Cells	3-7 x 10^6 cells	a549-ascov2-nlrp1
A549-ASC Cells (control)	3-7 x 10^6 cells	a549-ascg
A549-ASCoV2 Cells (control)	3-7 x 10^6 cells	a549-ascov2
Val-boroPro	10 mg	tlrl-vbp-10



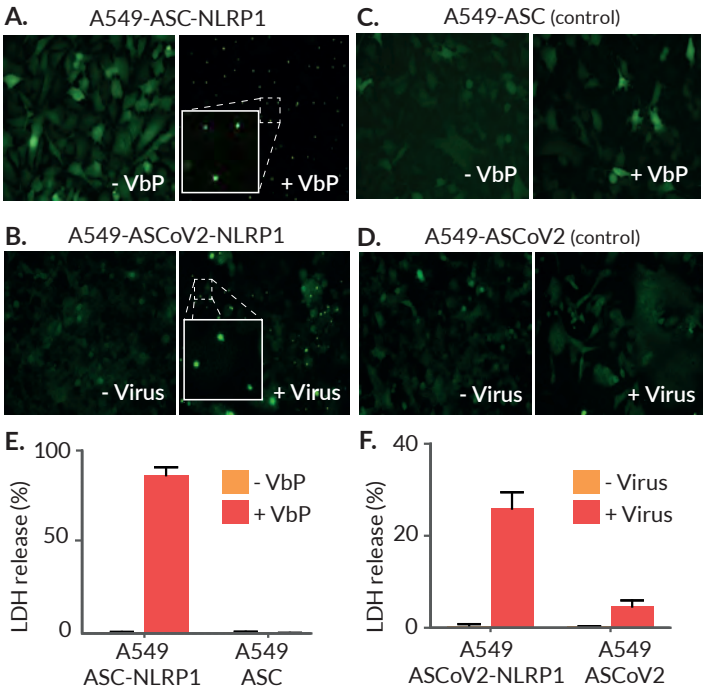


Figure 1. Monitoring of ASC::GFP speck formation and pyroptosis upon NLRP1 activation. All cell lines were incubated with the NFκB inducer hTNF-α ± Val-boroPro (VbP) **(A,C,E)** or ± SARS-CoV-2 viral particles (Virus) **(B,D,F)**. ASC::GFP expression and speck formation was monitored using fluorescence microscopy **(A-D)**. Subsequent pyroptosis was assessed by lactate dehydrogenase (LDH) assay. Data is shown in percentage of cell death **(E,F)**.

RELATED PRODUCTS

- **Recombinant human TNF-α:** recombinant cytokine (rcyc-htnfa)
- **Poly(I:C) (HMW):** Long synthetic analog of dsRNA (tlrl-pic)

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TLR4 Dual™ reporter cell lines

InvivoGen provides the first THP1-Dual™-derived reporter cell lines that allow the monitoring of the two major pathways induced by TLR4, the MyD88- and TRIF-dependent pathways. These cells overexpress MD-2 (myeloid differentiation factor 2) and CD14 (cluster of differentiation 14) to restore a functional TRIF-dependent response, and also TLR4 to increase their sensitivity to smooth and rough LPS (lipopolysaccharide). Thus, the cells can be used for detection of LPS, which represents a major threat for health, research and industry.

- **THP1-Dual™**
- **THP1-Dual™ MD2-CD14-TLR4** NEW
- **THP1-Dual™ MD2-CD14 KO-TLR4 (control)** NEW

Key Features

- Dual reporter system: IRF-Lucia and NF-κB-SEAP
- Restoration of the TRIF-dependent pathway
- No need for PMA differentation
- Highly responsive to smooth (s) and rough (r) LPS

THP-1 monocytes offer a physiological background for innate immunity studies as they express various PRRs (e.g. TLR4) as well as TLR-related genes involved in the signaling cascade[1]. InvivoGen's **THP1-Dual™** cells feature two inducible reporter genes for Lucia luciferase and SEAP (secreted embryonic alkaline phosphatase) to monitor the TRIF-dependent IRF and MyD88-dependent NF-κB pathway, respectively.

In **THP1-Dual™ MD2-CD14-TLR4 cells**, the IRF response to rLPS and sLPS is restored due to the stable expression of MD-2, CD14 and TLR4 *(Fig. 1)*. This feature eliminates the need for time consuming PMA-differentiation of the cells. In addition, the NF-κB response upon LPS stimulation is significantly increased *(Fig. 2)*.

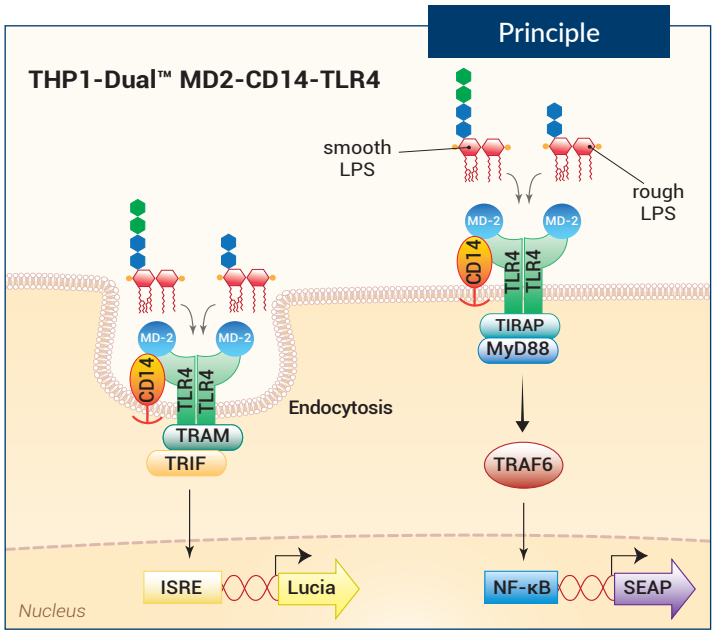
The **THP1-Dual™ MD2-CD14 KO-TLR4** control cells were engineered through a biallelic KO of TLR4 and do not respond to any type of LPS (Fig. 1&2).

InvivoGen also offers convenient antimicrobial reagents and sensitive endotoxin detection kit. Check our website to learn more. www.invivogen.com/endotoxin-detection-kit

PRODUCTS	QTY	CAT. CODE
THP1-Dual™ Cells	3-7 x 10^6 cells	thpd
THP1-Dual™ MD2-CD14-TLR4 Cells	3-7 x 10^6 cells	thpd-mctlr4
THP1-Dual™ MD2-CD14 KO-TLR4 Cells	3-7 x 10^6 cells	thpd-mckotlr4

RELATED PRODUCTS

- **LPS-EK Ultrapure:** Rough LPS from *E. coli* K12 (tlrl-peklps)
- **LPS-EB Ultrapure:** Smooth LPS from *E. coli* 0111:B4 (tlrl-pelps)
- **QUANTI-Blue™ Solution:** SEAP detection reagent, liquid (rep-qbs)
- **QUANTI-Luc™:** Luciferase detection reagent, powder (rep-qlc1)



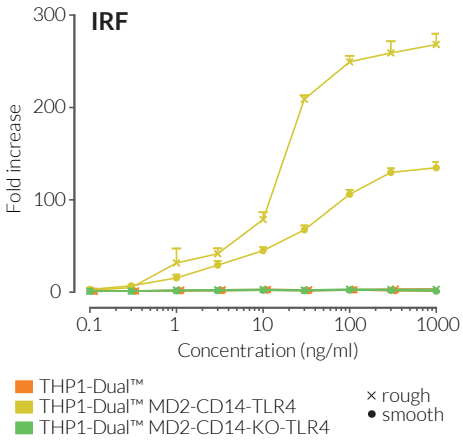


Figure 1. IRF responses to rough and smooth LPS in THP1-Dual™-derived cells. Cells were incubated for 24 hours with increasing concentration of rough (LPS-EK ultrapure (UP)) and smooth (LPS-EB UP) LPS. The IRF response was assessed by measuring the activity of Lucia luciferase in the supernatant using QUANTI-Luc™. Data are shown in fold response over non-induced cells (mean ± SEM).

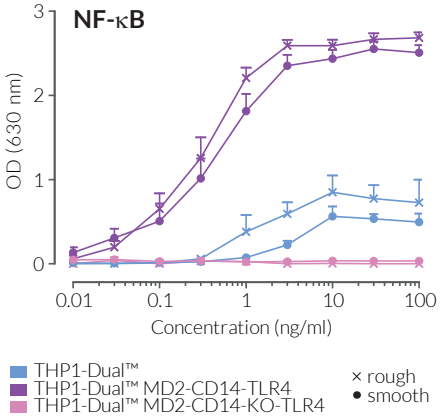


Figure 2. NF-κB responses to rough and smooth LPS in THP1-Dual™ -derived cells. Cells were incubated for 24 hours with increasing concentration of rough (LPS-EK UP) and smooth (LPS-EB UP) LPS. The activation of NF-κB was assessed by measuring the activity of SEAP in the supernatant using QUANTI-Blue™ Solution. Data are shown as optical density (OD) at 630 nm (mean ± SEM).

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QUANTI-Luc™ 4 Lucia/Gaussia

InvivoGen offers a liquid formulation of QUANTI-Luc™, a ready-to-use reagent designed for the bioluminescent detection of Lucia and Gaussia, two coelenterazine-utilizing secreted luciferases. QUANTI-Luc™ 4 Lucia/Gaussia is adapted for Flash or Glow luminescence detection in live-cell supernatants. Ultimately, this new formulation will replace QUANTI-Luc™ (powder) and QUANTI-Luc™ Gold (powder).

A Secreted Luciferase Detection Reagent NEW

QUANTI-Luc™ 4 Lucia/Gaussia is a liquid formulation of InvivoGen's highly cited QUANTI-Luc™ (Gold) detection reagent. It is designed for sensitive detection of secreted **Lucia** or **Gaussia** activity in live-cell supernatants, and intracellular Renilla after cell lysis.
For a more convenient use, this product allows extemporaneous dilution and Flash or Glow detection.

QUANTI-Luc™ 4 Lucia/Gaussia comprises two liquid components:

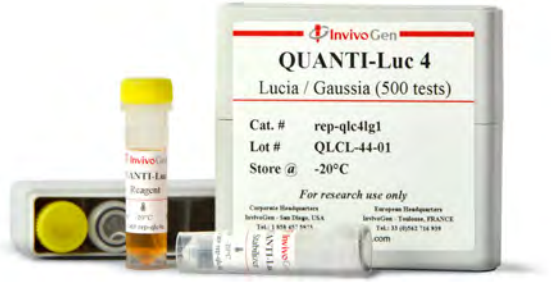
- QUANTI-Luc™ 4 Reagent 20X (coelenterazine substrate)
- QUANTI-Luc™ 4 Stabilizer 25X (optimized Glow assay reagent)

It provides a bright signal for both Flash and Glow luciferase detection:

- Flash detection of luciferase activity only requires mixing of cell supernatant (or lysate) with QUANTI-Luc™ 4 Reagent.
- Glow detection of luciferase activity requires simple addition of QUANTI-Luc™ 4 Stabilizer to the mix for enhanced signal stability.

Key Features

- Convenient, stable, and cost-effective
- Optimized for Lucia and Gaussia secreted luciferases
- Compatible with Renilla luciferase in cell lysates
- Suitable for Flash and Glow detection
- Compatible with dual reporter detection (e.g. Lucia & SEAP)



PRODUCTS	QTY	CAT. CODE
	500 tests	rep-qlc4lg1
QUANTI-Luc™ 4 Lucia/Gaussia	2 x 500 tests	rep-qlc4lg2
	5 x 500 tests	rep-qlc4lg5

RELATED PRODUCTS

- **QUANTI-Luc™:** Flash detection reagent, powder (rep-qlc1)
- **QUANTI-Luc™ Gold:** Flash and Glow detection reagent, powder (rep-qlcg1)
- **Lucia reporter cell line collection:** invivogen.com/cell-lines

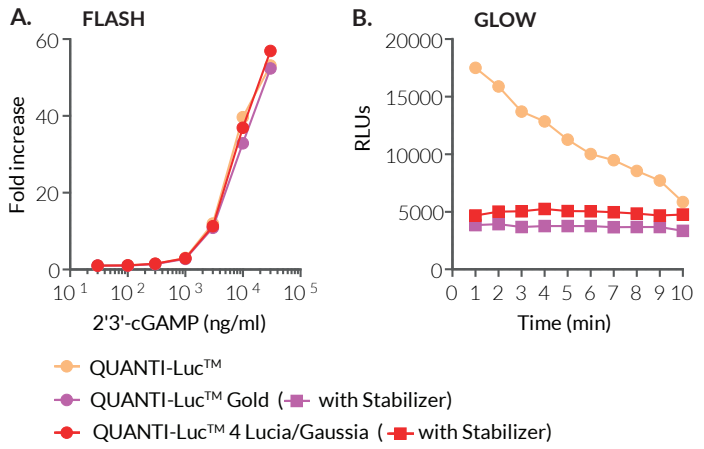


Figure 1. Lucia detection using QUANTI-Luc™, QUANTI-Luc™ Gold, and QUANTI-Luc™ 4 Lucia/Gaussia. THP1-Dual™ cells, which feature an IRF-inducible Lucia reporter gene, were cultured with increasing concentration of 2'3'-cGAMP, a STING agonist. After overnight incubation, QUANTI-Luc™, QUANTI-Luc™ Gold, or QUANTI-Luc™ 4 Lucia/Gaussia were added to the supernatants. **(A)** For Flash detection, Lucia activity was assessed immediately. Data is shown as fold increase. **(B)** For Glow detection, the Stabilizer was added to QUANTI-Luc™ Gold and QUANTI-Luc™ 4 Lucia/Gaussia prior to Lucia activity measurement every minute for 10 minutes. Data is shown as relative light units (RLUs).

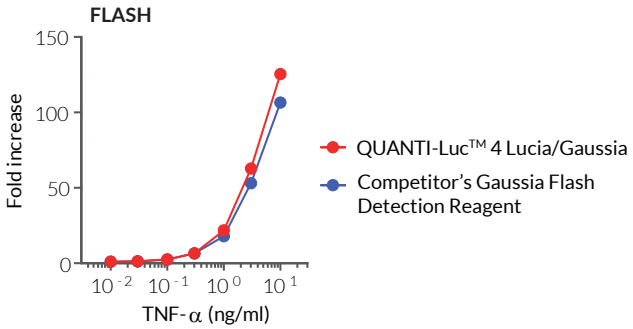


Figure 2. Flash detection of Gaussia luciferase. HEK293 cells stably transfected with an NF-κB-inducible Gaussia reporter gene were cultured with increasing concentration of human TNF-α. After overnight incubation, the Gaussia activity in the supernatant was assessed immediately after addition of InvivoGen's QUANTI-Luc™ 4 Lucia/Gaussia or a Gaussia detection reagent from a competitor. Data is shown as relative light units (RLUs).

... STAY TUNED !

QUANTI-Luc™ 4 Renilla will be available in Spring 2023!
This liquid formulation comprises a Lysis Buffer allowing the detection of the intracellular coelenterazine-based Renilla luciferase.

QUANTI-Luc™ (powder) and QUANTI-Luc™ Gold (powder) will be discontinued in June, 2023.

www.invivogen.com/quanti-luc

Mycoplasma Detection With MycoStrip™

With over 40 years of experience in developing mycoplasma solutions for the scientific community, InvivoGen now offers MycoStrip™, a simple strip-based test that requires no special lab equipment for the detection of mycoplasma in cell culture. Importantly, with results in less than an hour, MycoStrip™ allows you to swiftly combat the contamination.

Mycoplasma Detection Kit

- **MycoStrip™ 10/20/50 tests**
- **MycoStrip™ 50 tests (without cassettes)** NEW
- **MycoStrip™ 100 tests (without cassettes)**

Key Features

- **Rapid:** performed in 1 hour, <15 min hands-on-time
- **Clear:** results easy-to-interpret
  - One band - negative for mycoplasma
  - Two bands - positive for mycoplasma
- **Fast results:** within 2-5 min
- **Specific:** detects the six species that account for 95% of all contaminations. No cross-reactivity with other bacterial, fungal and mammalian DNA.
- **Sensitive:** able to detect as low as 10-10^2 CFU/ml
- **Available with cassettes:** convenient use
- **Available without cassettes:** economical and eco-friendly

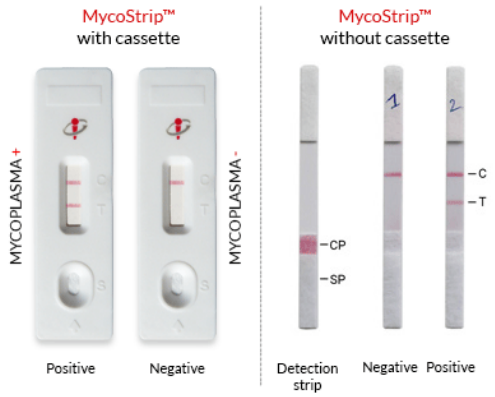
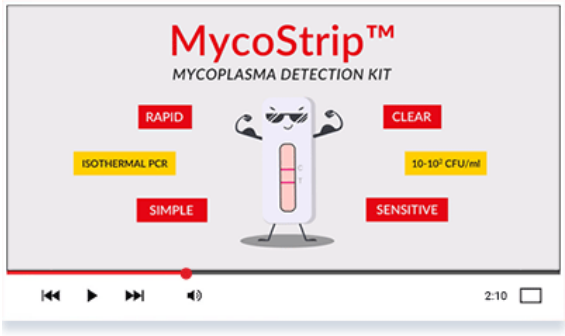


Figure 1. Mycoplasma detection using MycoStrip™ with or without cassette.
SP: Sample Pad, CP: Conjugation Pad, C: Positive Control Band, T: Test Band.

Watch our video on MycoStrip™



PRODUCTS	QTY	CAT. CODE
MycoStrip™	10 tests	rep-mys-10
	20 tests	rep-mys-20
	50 tests	rep-mys-50
MycoStrip™ 50	50 tests (without cassettes)	rep-mysnc-50
MycoStrip™ 100	100 tests (without cassettes)	rep-mysnc-100

RELATED PRODUCTS

- **Plasmocin®:** Mycoplasma elimination reagent (ant-mpp)
- **Plasmocure™:** Alternate reagent for mycoplasma elimination (ant-pc)
- **Normocin™:** Anti-microbial reagent for preventing contamination of cell cultures by mycoplasmas, bacteria or fungi (ant-nr1)
- **PlasmoTest™:** Cell-based assay for mycoplasma detection (rep-pt1)

FREQUENTLY ASKED QUESTIONS

Q: What if I don't see the positive control band ?
A: The absence of positive control band on the strip means that the reaction failed. MycoStrip™ is based on isothermal PCR for which the optimal temperature is 65°C. We strongly recommend that you use a thermocycler, or check the temperature of the heat-block with a thermometer.

Q: What if my test is positive for mycoplasma?
A: InvivoGen has the solution! Your culture is easily treatable with our comprehensive anti-mycoplasma reagents. Simply treat your culture with Plasmocin® or Plasmocure™ and upon completion of the treatment (~2 weeks), re-test using MycoStrip™.

Q: When should I test for Mycoplasma contamination?
A: We strongly recommend testing when you receive cells from another source/lab, when you notice a change in cell growth and/or morphology or on a routine basis (e.g. every ~2-3 weeks).

Q: How can I prevent mycoplasma contamination?
A: InvivoGen offers multiple antimicrobial reagents to prevent Mycoplasma contamination : Normocin™, Plasmocin® prophylactic and Primocin® depending on your cell type.

www.invivogen.com/mycostrip

InvivoGames: Brain stimulation assay

Crossword puzzle

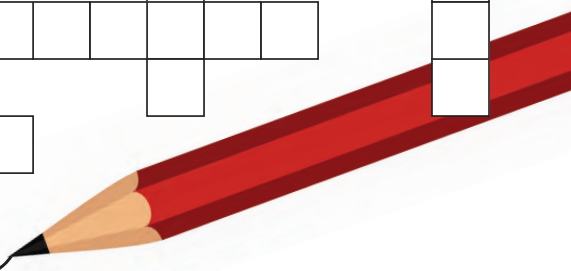
GRAB A PEN AND CHALLENGE YOURSELF!

Across

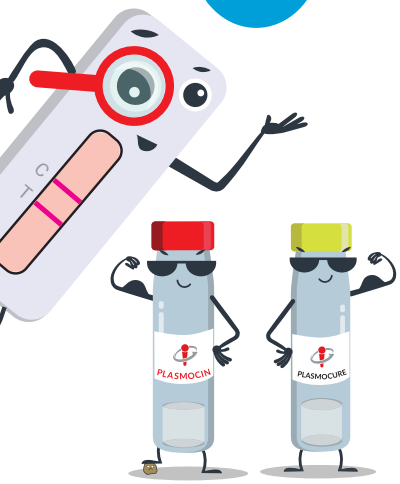
3. It can be rough or smooth
6. A pore-forming protein
7. Specific domain in NLRP1
11. Meant to bind
12. Immune cell messengers
13. Defense mechanism
15. InvivoGen's luciferase

Down

1. Protein scissor
2. Antibiotic by InvivoGen
4. Cell death through fire
5. Degradation complex of the cell
8. Inflammasome with a pyrin domain
9. Useful to track cells in the dark
10. The T in TLR stands for?
14. Inflammasome player



DON'T LOSE YOUR HEAD OVER MYCOPLASMA! USE PLASMOCURE™ & PLASMOCIN®



www.invivogen.com/mycoplasma-elimination

Find 10 differences

Solution (top to bottom, left to right): nunchucks, grenade, scull, finger, mycoplasma: in pocket, behind bottle, tube missing, mycoplasma inside bottle (2x), mycoplasma missing

Solution: 1.Caspase 2.Zeocin 3.LPS 4.Pyroptosis 5.Proteasome 6.Gasdermin 7.FIIND 8.NLRP 9.GFP 10.Toll 11.Receptor 12.Cytokines 13.Inflammation 14.ASC 15.Lucia

InvivoGen

Europe	Tel: +33 562 71 69 39	Fax: +33 562 71 69 30	info.eu@invivogen.com
USA	Tel: +1 888 457 5873	Fax: +1 858 457 5843	info@invivogen.com
Asia	Tel: +852 3622 3480	Fax: +852 3622 3483	info.hk@invivogen.com