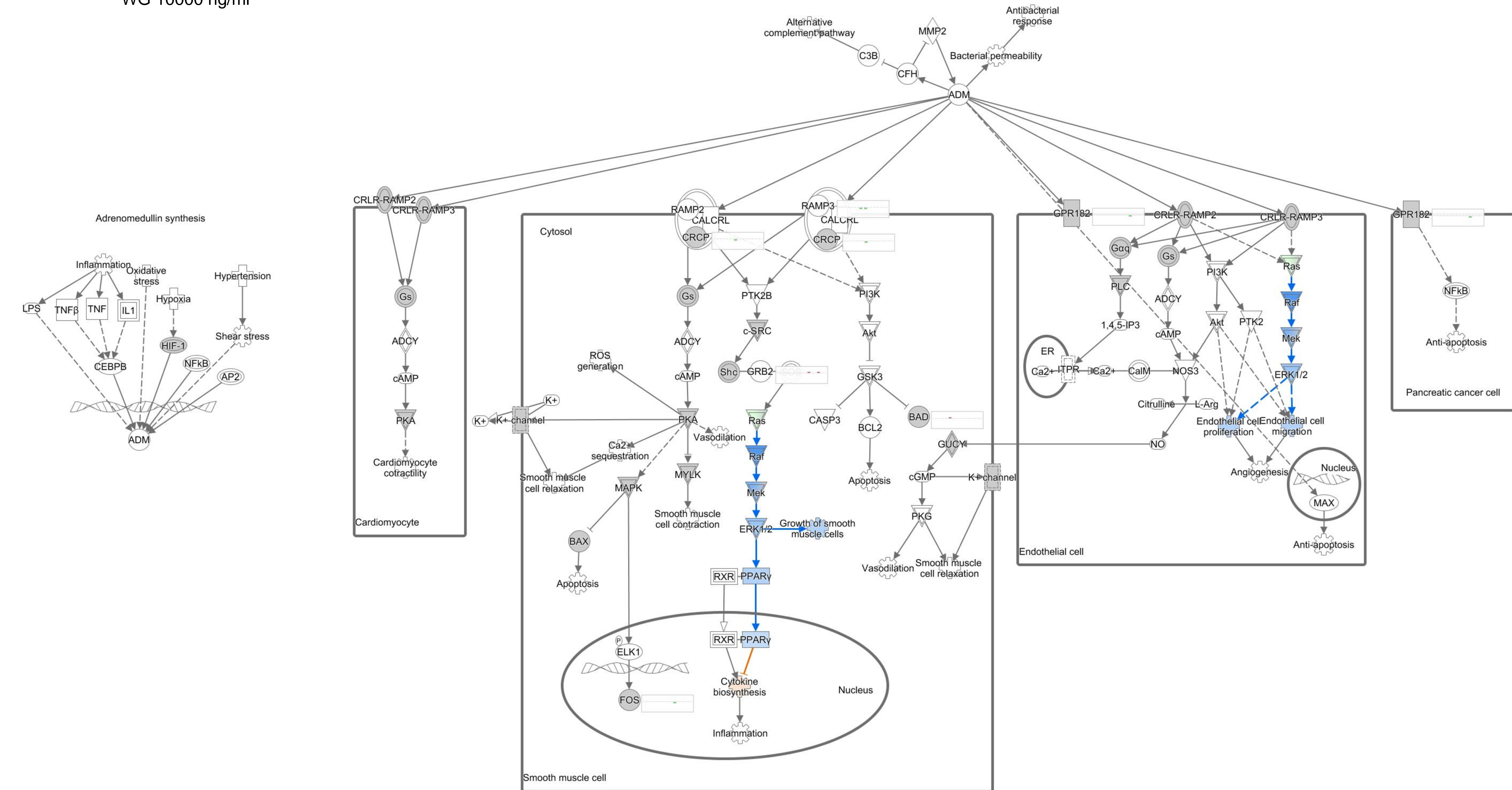


Adrenomedullin (AM) is a member of the calcitonin gene-related peptide (CGRP) family. It is produced by many different cell types throughout the body, with the possible exception of the thyroid and thymus, and has vasodilatory, hypotensive and angiogenic activity. AM also stimulates cell growth and inhibits apoptosis in many tumor cells.

WG 10000 ng/ml



HRG80 10000 ng/ml

The diagram illustrates the signaling pathways and cellular effects of HRG80 (10000 ng/ml) across four cell types: Cardiomycocyte, Smooth muscle cell, Endothelial cell, and Pancreatic cancer cell.

Central Signaling Hub: ADM (Adrenomedullin) is the central molecule, interacting with various receptors and signaling pathways. Key receptors include CRLR-RAMP2, CRLR-RAMP3, GPR182, and Gs. ADM also interacts with the alternative complement pathway (C3B, CFH, MMP2) and bacterial permeability.

Cardiomycocyte Pathway: ADM binds to CRLR-RAMP2 and CRLR-RAMP3, activating Gs. This leads to ADCY, cAMP, and PKA, resulting in Cardiomycocyte contractility. ADM also interacts with CRLR-RAMP3, leading to Gs, ADCY, cAMP, and PKA, resulting in Cardiomycocyte contractility.

Smooth muscle cell Pathway: ADM binds to RAMP2, RAMP3, and Gs. RAMP2 and RAMP3 activate Gs, leading to ADCY, cAMP, and PKA. PKA leads to smooth muscle cell relaxation (via K⁺ channel) and smooth muscle cell contraction (via MYLK). RAMP3 also activates PI3K, leading to Akt, BCL2, and Apoptosis. RAMP3 also activates Ras, leading to Raf, Mek, and ERK1/2, resulting in Growth of smooth muscle cells. ADM also interacts with Gs, leading to ADCY, cAMP, and PKA, resulting in smooth muscle cell relaxation (via K⁺ channel) and smooth muscle cell contraction (via MYLK). ADM also interacts with Gs, leading to ADCY, cAMP, and PKA, resulting in smooth muscle cell relaxation (via K⁺ channel) and smooth muscle cell contraction (via MYLK).

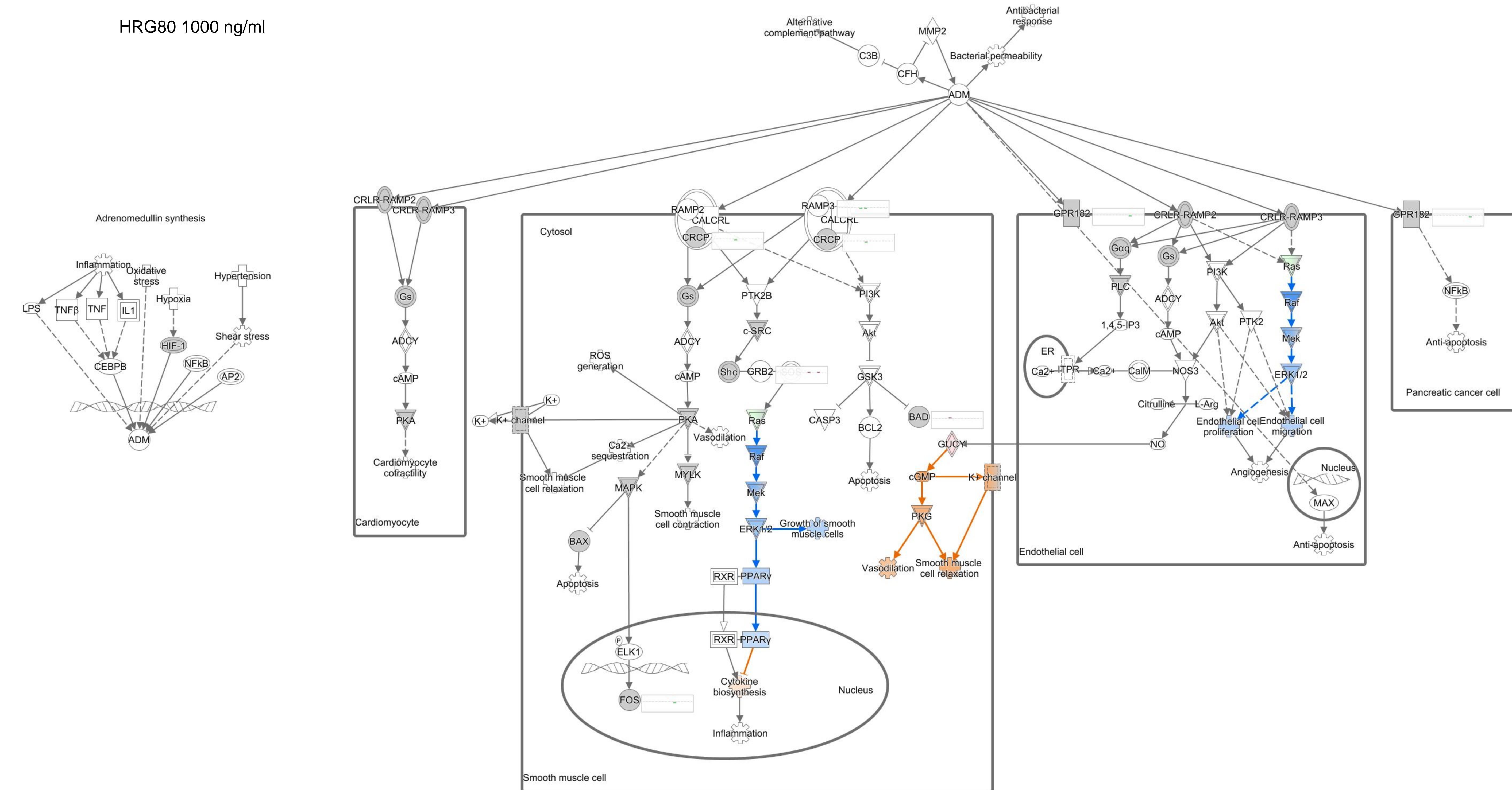
Endothelial cell Pathway: ADM binds to GPR182, CRLR-RAMP2, and CRLR-RAMP3. GPR182 activates Gq, leading to PLC, 1,4,5-IP3, and Ca²⁺. CRLR-RAMP2 and CRLR-RAMP3 activate Gs, leading to ADCY, cAMP, and PKA. PKA leads to smooth muscle cell relaxation (via K⁺ channel) and smooth muscle cell contraction (via MYLK). CRLR-RAMP3 also activates PI3K, leading to Akt, BCL2, and Apoptosis. CRLR-RAMP3 also activates Ras, leading to Raf, Mek, and ERK1/2, resulting in Growth of smooth muscle cells. ADM also interacts with Gs, leading to ADCY, cAMP, and PKA, resulting in smooth muscle cell relaxation (via K⁺ channel) and smooth muscle cell contraction (via MYLK). ADM also interacts with Gs, leading to ADCY, cAMP, and PKA, resulting in smooth muscle cell relaxation (via K⁺ channel) and smooth muscle cell contraction (via MYLK).

Pancreatic cancer cell Pathway: ADM binds to GPR182, leading to NFkB and Anti-apoptosis.

Other Pathways: ADM interacts with the alternative complement pathway (C3B, CFH, MMP2) and bacterial permeability. ADM also interacts with Gs, leading to ADCY, cAMP, and PKA, resulting in Cardiomycocyte contractility. ADM also interacts with Gs, leading to ADCY, cAMP, and PKA, resulting in Cardiomycocyte contractility.

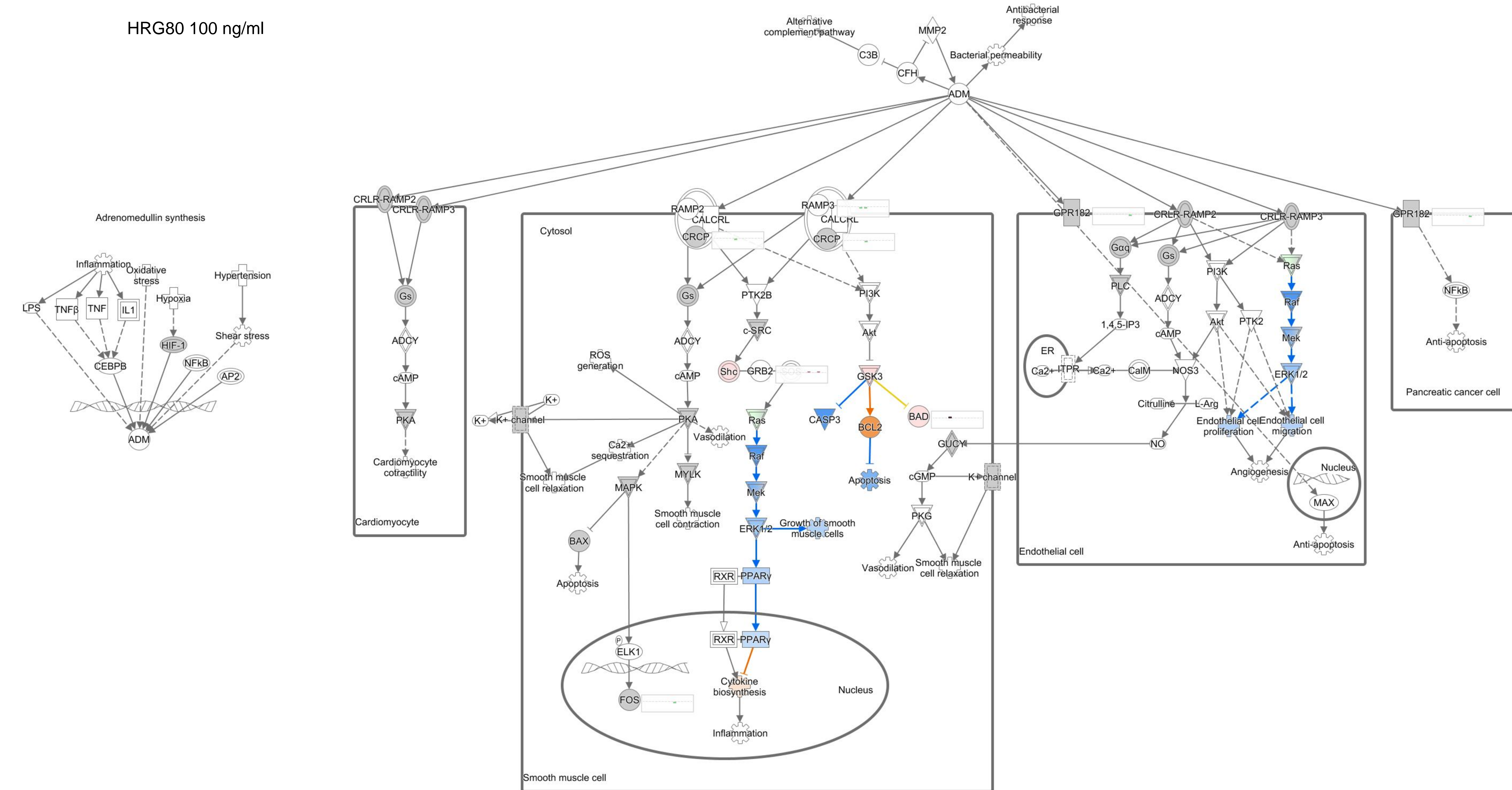
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HRG80 1000 ng/ml



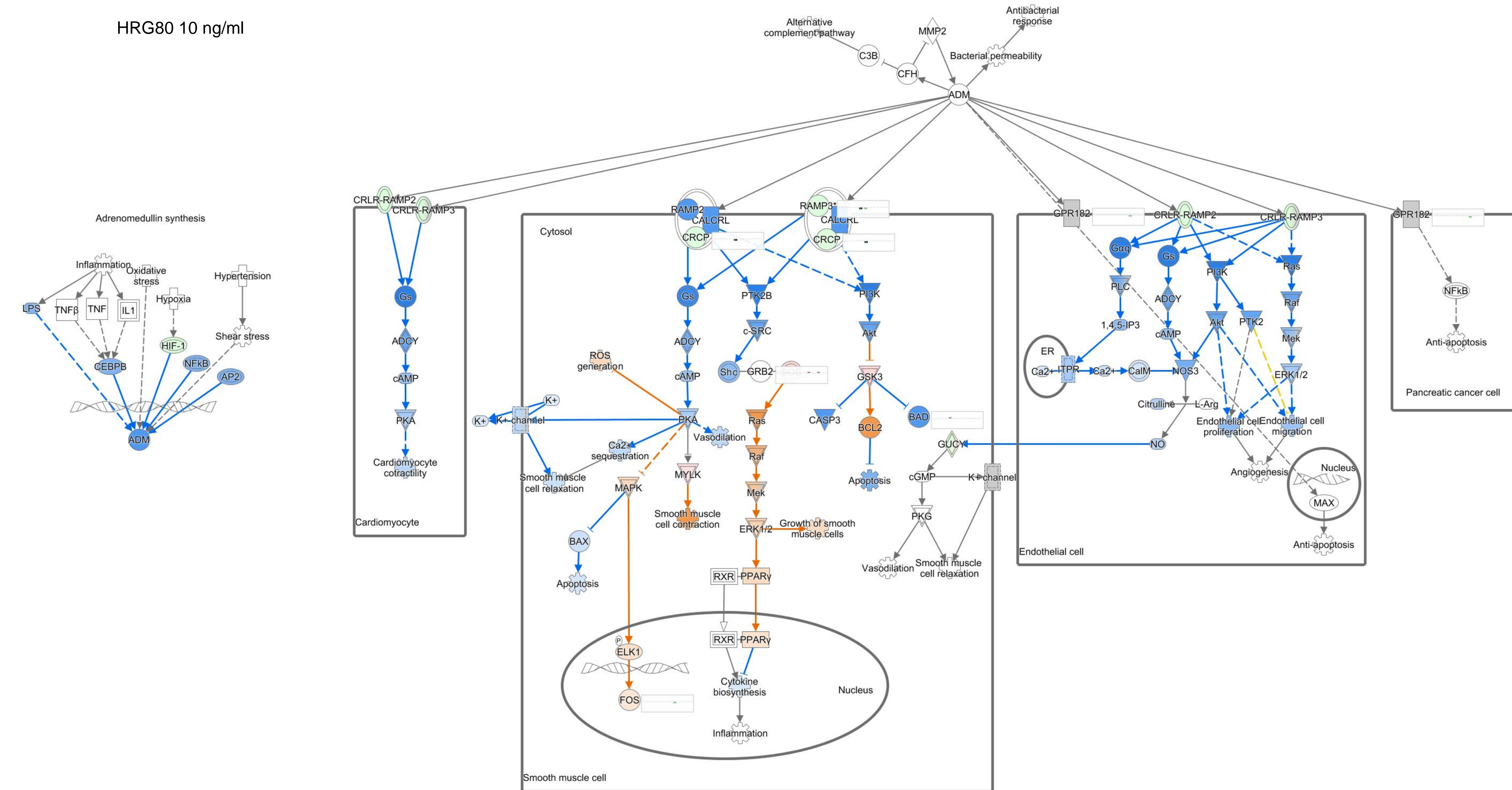
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HRG80 100 ng/ml



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HRG80 10 ng/ml



The diagram illustrates the regulation of Adrenomedullin (ADM) synthesis by various stimuli. At the top, five stimuli are shown: Inflammation (gear icon), Oxidative stress (square icon), Hypoxia (plus icon), Hypertension (plus icon), and Shear stress (gear icon). Inflammation leads to the activation of LPS (rounded rectangle), TNF α (square), TNF (square), and IL1 (square). Oxidative stress leads to the activation of CEBPB (oval). Hypoxia leads to the activation of HIF-1 (oval). Hypertension leads to the activation of Shear stress (gear icon). Shear stress leads to the activation of NF κ B (oval) and AP2 (oval). CEBPB, HIF-1, NF κ B, and AP2 all lead to the activation of ADM synthesis, represented by a DNA double helix and a shaded area labeled ADM at the bottom. Dashed lines indicate indirect or less direct pathways, while solid lines indicate direct pathways.



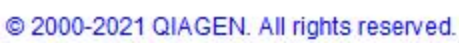
Rb1 100 nM

Adrenomedullin synthesis

The diagram illustrates the pathways leading to Adrenomedullin (ADM) synthesis. At the top, 'Rb1 100 nM' is indicated. Below it, 'Adrenomedullin synthesis' is the central theme. The pathways are as follows:

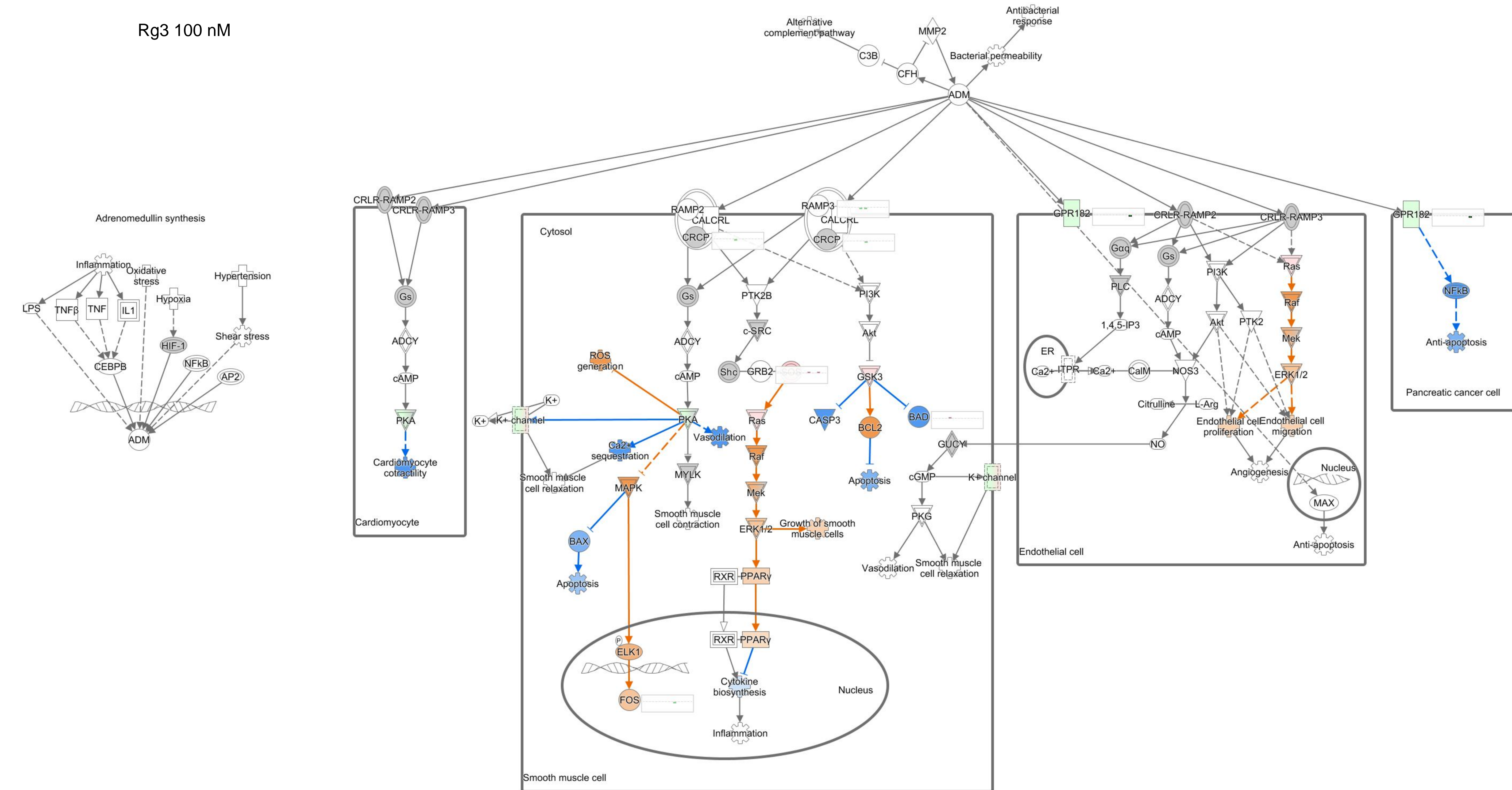
- Inflammation** (represented by a gear icon) leads to **LPS**, **TNF β** , **TNF**, and **IL1** (all in boxes).
- Oxidative stress** (represented by a plus sign icon) leads to **Hypoxia** (in a box).
- Hypertension** (represented by a plus sign icon) leads to **Shear stress** (represented by a gear icon).
- Shear stress** leads to **HIF-1** (in an oval).
- Hypoxia** leads to **HIF-1** (in an oval).
- LPS**, **TNF β** , **TNF**, and **IL1** all lead to **CEBPB** (in an oval).
- HIF-1** leads to **NFkB** (in an oval).
- Shear stress** leads to **AP2** (in an oval).
- CEBPB**, **HIF-1**, **NFkB**, and **AP2** all lead to **ADM** (in a box).

The final step is the synthesis of **ADM**, which is represented by a DNA double helix structure.



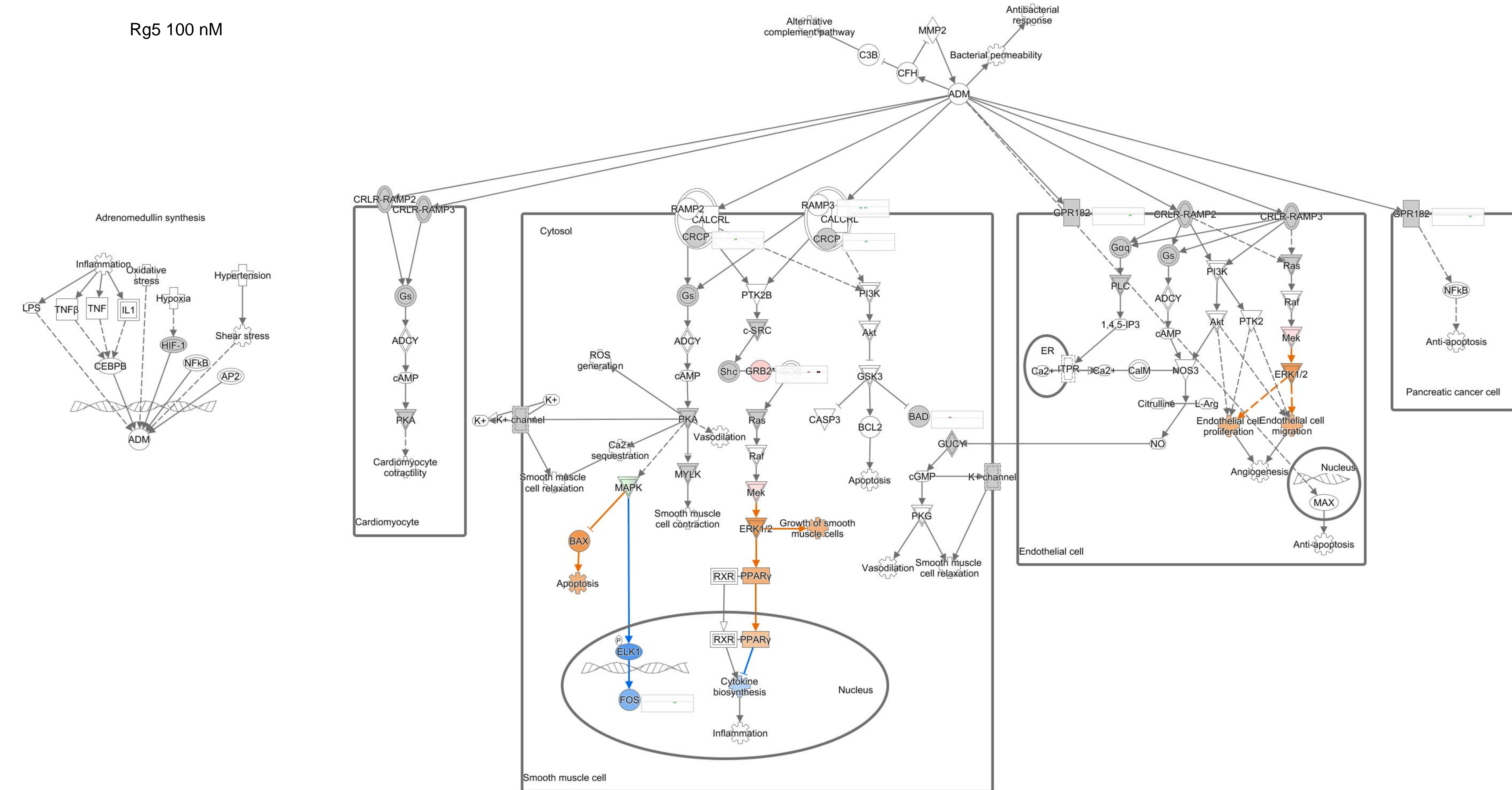
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Rg3 100 nM



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Rg5 100 nM



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Rk1 100 nM

