Making Them Remember
... my results and/or me

Bruce Schultz

VRSP
19 June, 2014

Outstanding in a Crowd

- Focus on …
  - Message
    - Succinct, Clear, Simple
  - Audience
    - Physical & academic characteristics
    - Questions – allow the data to speak
  - Presentation
    - Strategic & Practiced

Layout

Title

Authors

Affiliation

Abstract

Objectives

Methods 1

Results: Fig 1

Figure 1. Title.
Description

A

B

C

Figure 2. Title. A.
Description **B.**
Description **C.**

Methods 2

Fig 3 & legend

Fig 4 & legend

Fig 5 & legend

Fig 6 & legend

Conclusions: Drawing

Conclusions
Bullet list

Summary
Bullet list

Acknowledge-ments

Program #1039.13

Peroxisome Proliferator Receptor γ Agonists Alter Electrolyte Transport Across Porcine Vas Deferens Epithelia

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Kansas State University, Manhattan, KS 66506

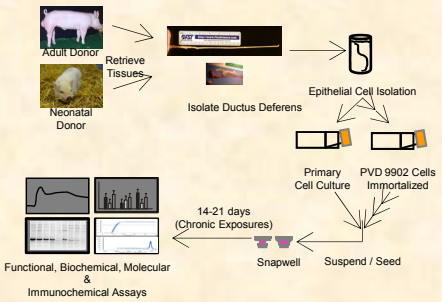
Abstract

Elevated levels of 15-deoxy-Δ12-14-prostaglandin-J2 (15dPGJ2) have been reported in the reproductive tracts of some cases of male infertility. The goal for this project was to determine a possible mechanism by which 15dPGJ2, an endogenous PPARγ ligand, might contribute to male infertility. Vas deferens epithelial cells were isolated from pigs, cultured for 14-21 days and exposed to dexamethasone and/or PPARγ agonists for the final 3-4 days of culture. Cells were mounted in modified Ussing chambers and exposed to amiloride (ENaC blocker), forskolin (adenylyl cyclase activator), and DASU-02 (CFTR blocker). Amiloride sensitive current induced by dexamethasone was potentiated two-fold by concurrent rosiglitazone exposure while there was no effect on baseline, forskolin or DASU-02 responses. Protein and RNA were isolated. Western blots suggest a decrease in CFTR expression and an increase in α, β, and γ ENaC subunits. RT-PCR detected a decrease in RNA coding for CFTR. PPARγ agonist treatment in the PVD9902 cell line attenuated forskolin and DASU-02 responses. These effects were concentration dependent, induced by structurally distinct PPARγ agonists, and blocked by a PPARγ antagonist, T0070907. These outcomes suggest that PPARγ activation by 15dPGJ2 in the reproductive duct could alter luminal electrolytes, which would likely affect sperm viability and function. [NIH R01-HD058398 & P20-RR017686 Core C]

Objectives

- To determine whether net ion transport across vas deferens epithelia is affected by PPARγ agonists in the absence or presence of dexamethasone.
- To determine whether the expression of ENaC and/or CFTR in vas deferens epithelial cells is affected by PPARγ agonist exposure.
- To test for PPARγ mediated modulation of vas deferens epithelial ion transport.

Experimental Procedures



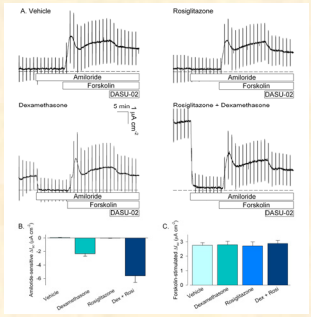


Figure 1. PPARγ agonist rosiglitazone enhances dexamethasone-induced amiloride-sensitive Na^+ absorption, but is without effect on forskolin-stimulated anion secretion across adult porcine vas deferens epithelial primary cell cultures. **A.)** Typical short circuit current (I_{sc}) results using epithelial cells isolated from a single pig vas deferens that were cultured in the absence or presence of rosiglitazone (10 μM), and dexamethasone (100 nM) as indicated for 4 days. Dashed lines indicate no net current. Results from panel A and eleven similar experiments are summarized in panels B & C. **B.)** Rosiglitazone causes a clear consistent potentiation of the dexamethasone-induced and amiloride-sensitive I_{sc}. Rosiglitazone, alone, was without effect on baseline I_{sc}. **C.)** Forskolin-stimulated anion secretion was unaffected by rosiglitazone exposure in culture.

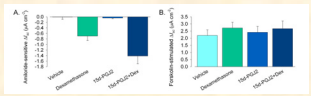


Figure 2. Endogenous PPARγ agonist 15d-PGJ2 enhances dexamethasone-induced amiloride-sensitive Na^+ absorption, but is without effect on forskolin-stimulated anion secretion across porcine vas deferens epithelial primary cell cultures. Vas deferens epithelia cells were isolated and cultured in the absence or presence of 15d-PGJ2 (10 μM) and/or dexamethasone (100 nM). Data are summarized from three experiments. **A.)** As with rosiglitazone exposure, 15d-PGJ2 potentiates amiloride-sensitive I_{sc} induced by dexamethasone while having no effect alone. **B.)** 15d-PGJ2 has no effect on forskolin-stimulated I_{sc}.

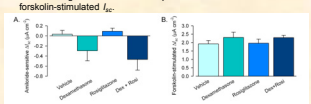


Figure 3. Amiloride-sensitive Na^+ absorption across epithelial cells isolated from neonatal vas deferens is enhanced by rosiglitazone exposure while anion secretion is unaffected. **A.)** As in adult primary porcine vas defrens cells, rosiglitazone (10 μM) potentiated the effect of dexamethasone (100 nM) on amiloride-sensitive Na^+ absorption (compare to Fig. 1B). **B.)** The magnitude of forskolin-stimulated anion secretion was not affected by exposure to either rosiglitazone or dexamethasone. Data are summarized from eight experiments.

Results

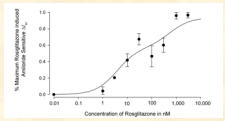


Figure 4. Rosiglitazone potentiated amiloride sensitive I_{sc} in a concentration dependent manner. Cells were cultured in the absence or presence of selected rosiglitazone concentrations for four days and mounted in a modified Ussing chamber. The solid line represents the best fit of a modified Hill equation to the data set. The curve suggests a two part process with apparent K_Ds of 38 nM and 524 nM. These data are consistent with rosiglitazone activating PPARγ with a K_D of 38 nM and one or more of its isoforms- PPARα/β/δ with a K_D of 524 nM.

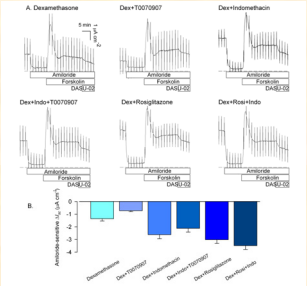


Figure 5. PPARγ antagonist T0070907 inhibits amiloride-sensitive dexamethasone induced I_{sc}. Cox-02 inhibitor indomethacin failed to exhibit the same effect but rather, increased amiloride sensitive I_{sc}, an effect which appears to be independent of PPARγ. **A.)** Typical results using epithelial cells isolated from a single pig vas deferens that were cultured in the absence or presence of dexameth-asone (100 nM), T0070907 (100 nM), indomethacin (50 μM), and/or Rosiglitazone (3 μM) as indicated, for 4 days. Five similar experiments are summarized in panel B. **B.)** T0070907 causes a clear consistent attenuation of the dexamethasone-induced and amiloride-sensitive I_{sc}. Indomethacin did not mimic this effect but rather caused a consistent increase in the amiloride sensitive I_{sc}. Results of T0070907 and/or rosiglitazone treatment in combination with indomethacin suggests that this effect is independent of PPARγ.

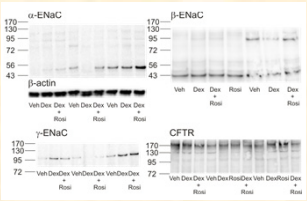


Figure 6. Indirect immunofluorescence suggests increased expression of α, β, and γ ENaC subunits with lowered expression of CFTR in cells exposed to rosiglitazone. Initial observations by western blot show increased signals for α, β, and γ ENaC and a decreased signal for CFTR following exposure to rosiglitazone. Cells isolated from three animals were cultured as monolayers in vehicle (Veh), dexamethasone (Dex; 100 nM), or Dex plus rosiglitazone (10 μM; Dex+Rosi). Cell lysates were resolved by SDS-PAGE and probed with antibodies to epitopes of the indicated proteins. All outcomes are shown.

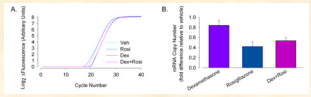


Figure 7. RT-PCR shows approximately 50% less mRNA coding for CFTR in PVD cells exposed to rosiglitazone. PVD cell monolayers were cultured in the presence of vehicle, dexamethasone (100 nM; Dex), rosiglitazone (10 μM; Rosi), or Dex+Rosi. **A.)** Product amplification from a single experiment in which RNA was probed with primers to detect CFTR. A 1 cycle rightward shift is observed following exposure to rosiglitazone. **B.)** Results summarized from three experiments showing a decrease in mRNA coding for CFTR in cells exposed to rosiglitazone. Results were normalized to the amplification of 18S RNA (ΔΔCt).

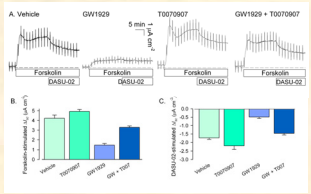


Figure 8. Inhibition of forskolin-stimulated anion secretion across PVD9902 cell monolayers by GW1929, a PPARγ agonist, is blocked by T0070907, a PPARγ antagonist. PVD9902 cells were cultured in the absence or presence of GW1929 (100 nM) and/or T0070907 (100 nM). **A.)** Typical results from a single experiment. **B. & C.)** Results summarized from panel A and three similar experiments. The I_{sc} responses to forskolin and DASU-02 were attenuated by GW1929. T0070907 did not affect baseline I_{sc} or the response to forskolin, but substantially prevented the inhibitory effect of GW1929.

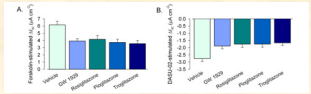


Figure 9. Four PPARγ agonists equally attenuated the forskolin-stimulated I_{sc} across PVD9902 epithelial cell monolayers. PVD9902 cells were cultured with vehicle, GW1929 (100 nM), rosiglitazone (10 μM), pioglitazone (10 μM), or troglitazone (10 μM) for 2-4 days, as indicated. **A.)** All the PPARγ agonists reduced the I_{sc} response to forskolin by a similar magnitude. **B.)** The magnitude of DASU-02 inhibition was reduced proportionately by the PPARγ agonists. Data are summarized from five experiments.

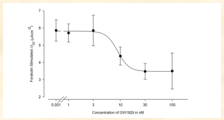
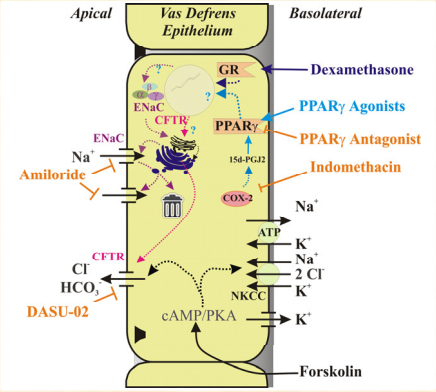


Figure 10. GW 1929 attenuates forskolin-induced I_{sc} across PVD9902 monolayers in a concentration dependent manner. PVD9902 cells were cultured in the absence or presence of selected GW 1929 concentrations for three days and mounted in a modified Ussing chamber. The solid line represent the fit of a modified Hill equation to the data set. Data were well-fitted with a Hill coefficient of one. An apparent K_D of 8.7 nM was derived. Data are summarized from 7 experiments.

Conclusions



- A subset of cellular components that can account for ion transport across cultured monolayers of porcine vas deferens epithelial cells is depicted.
- PPARγ is thought to be a nuclear receptor that modulates gene expression.
- Dashed arrows indicated pathways that are potentially activated or affected by PPARγ, either directly or indirectly. PPARγ may be a direct co-modulator for the expression of distinct ion transport proteins (i.e., upregulate ENaC expression and downregulate CFTR expression) or it might affect the expression of signaling pathway or protein trafficking components (e.g., SGK or ubiquitin) that ultimately modulate ion transport.

Summary

- PPARγ agonists enhance the effect of dexamethasone on I_{sc} while having no effect on I_{sc} alone in adult and neonatal primary vas deferens epithelia cells.
- Initial observations by western blot suggest increased ENaC expression and decreased CFTR expression.
- mRNA coding for CFTR is decreased in cells exposed to rosiglitazone.
- PPARγ agonists decrease the I_{sc} response by PVD9902 cells to forskolin and DASU-02.
- The PPARγ antagonist T0070907 partially blocked the effect of GW1929 on PVD 9902 cells.
- GW1929 exhibited concentration dependent attenuation of forskolin-stimulated current.

Special thanks to…

Sheng Yi
Jimmie Stewart
Dr. Fernando Pierucci-Alves

Support

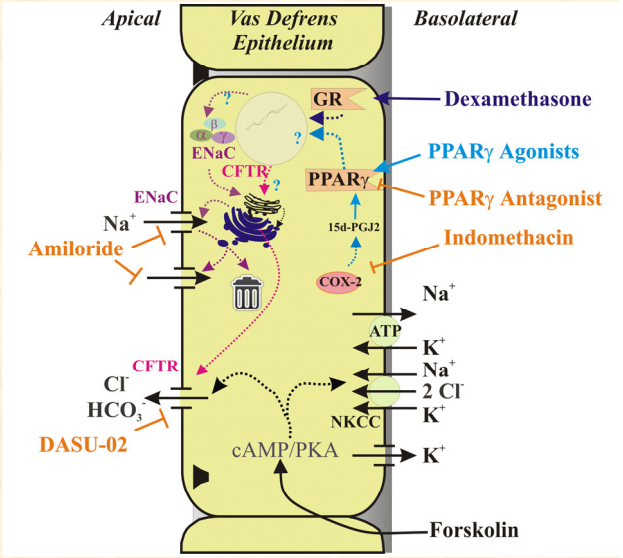
NIH R01-HD058398
P20-RR017686 Core C

Title: the Punch Line

- Provides a "take-home" message
- Includes key words
- Provides context
  - Species, tissue, cell type, etc.
- Is focused
  - Less than 100 characters
- May be the only information that is read

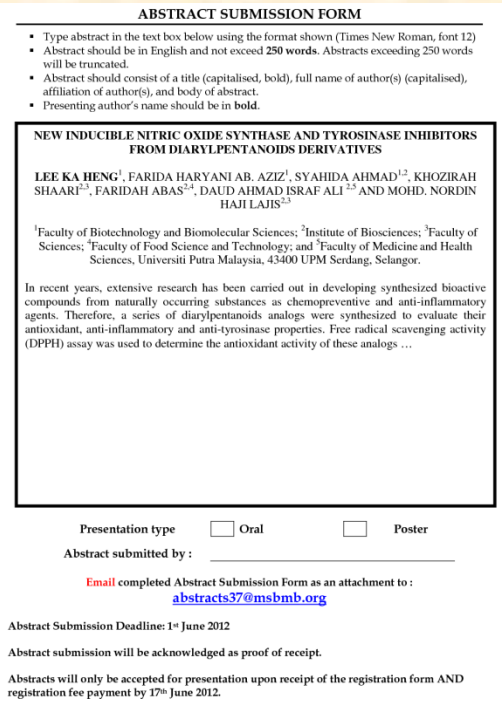
Conclusions:
the most important image

- Interpretation of your observations placed in relevant context
- Drawing & bullet points
- Simple, but complete



Abstract

- Submitted document verbatim
- Thought, creative work, and word-smithing completed prior to submission
- Focused and complete
  - All authors must approve
  - NO PROMISES!



ABSTRACT SUBMISSION FORM

- Type abstract in the text box below using the format shown (Times New Roman, font 12)
- Abstract should be in English and not exceed **250 words**. Abstracts exceeding 250 words will be truncated.
- Abstract should consist of a title (capitalised, bold), full name of author(s) (capitalised), affiliation of author(s), and body of abstract.
- Presenting author's name should be in **bold**.

NEW INDUCIBLE NITRIC OXIDE SYNTHASE AND TYROSINASE INHIBITORS FROM DIARYLPENTANOIDS DERIVATIVES

LEE KA HENG[1], FARIDA HARYANI AB. AZIZ[1], SYAHIDA AHMAD[1,2], KHOZIRAH SHAARI[2,3], FARIDAH ABAS[2,4], DAUD AHMAD ISRAF ALI [2,5] AND MOHD. NORDIN HAJI LAJIS[2,3]

[1]Faculty of Biotechnology and Biomolecular Sciences; [2]Institute of Biosciences; [3]Faculty of Sciences; [4]Faculty of Food Science and Technology; and [5]Faculty of Medicine and Health Sciences, Universiti Putra Malaysia, 43400 UPM Serdang, Selangor.

In recent years, extensive research has been carried out in developing synthesized bioactive compounds from naturally occurring substances as chemopreventive and anti-inflammatory agents. Therefore, a series of diarylpentanoids analogs were synthesized to evaluate their antioxidant, anti-inflammatory and anti-tyrosinase properties. Free radical scavenging activity (DPPH) assay was used to determine the antioxidant activity of these analogs …

Presentation type ☐ **Oral** ☐ **Poster**

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Abstract Submission Deadline: 1st June 2012

Abstract submission will be acknowledged as proof of receipt.

Abstracts will only be accepted for presentation upon receipt of the registration form AND registration fee payment by 17th June 2012.

Results:
foundation of conclusions

- Well-labeled and uncluttered
- Prepared for this presentation
- NOT …
  - Pixelated, stretched, distorted
- Complete, but concise legends
  - Title is summary
  - Tell the reader what they should see

Summary

- Bring key points together
- One bullet per figure
- Start with figure titles

Methods - minimal

- Diagrammatic presentation
- Flow chart
- Typical outcomes shown or described
- Used in presentation **only** if directly requested or required

Background Information

- Not required
  - Keep to a minimum
- Objective(s)
- Hypothesis?
- Bullet points

- Focus on **your** message, the new knowledge that you discovered

Acknowledgements

- Critical contributors
  - Technical support, supplier of critical reagents, etc.
  - Affiliation (if other than the authors)
  - No co-authors

- Funding sources
  - NIH(T35OD010979)
  - Benjamin Kurz Scholarship
  - Merial Animal Health
  - Morris Animal Foundation
  - K-State CVM
  - Zoetis
  - BRI (Biosecurity Research Institute)

Layout – make it easy to get the message

- Effective use of drawings, graphs and images
  - Every picture tells a story – insure that it is your story
- Readable font and size
- Effective use of colors
- Vanishing background
- Pay attention to detail!

Layout

Title

Authors

Affiliation

Abstract

Objectives

Methods 1

Results: Fig 1

Figure 1. Title.
Description

A

B

C

Figure 2. Title. A.
Description **B.**
Description **C.**

Methods 2

Fig 3 & legend

Fig 4 & legend

Fig 5 & legend

Fig 6 & legend

Conclusions: Drawing

Conclusions
Bullet list

Summary
Bullet list

Acknowledge-ments

Presentation: Start with the 'take-home' message

- Message is well-focused in 'Conclusions' – start here
- Walk through conclusions in logical sequence
  - point to supporting data
- Include only salient methods
- Listen closely to questions
  - Answer questions directly

Preparation: an iterative, mentored, practiced process

- Start early
- Look at good examples
- Work with your mentor
- Seek questions from peers
- Practice, practice, practice

Help the Audience Remember

- Title = take-home message
- Conclusions – clear & relevant
- Layout – logical and readable
  - Pay attention to detail
- Presentation – crisp
  - Preparation and practice


NHG ASC 2009

Best Poster Presentation Award

Category	Winner	Institution
Allied Health	Mr Gary Lee Jek Chong	Temasek Poly
Basic Science	Ms Quah Phaik Ling	NUHS
Primary Care	Dr Matthias Toh	NHGP
Medical Disciplines	Dr Effie Chew	NUHS
Nursing	Ms Chui Kui Lin, Winnie	AH
QHSR	Ms Wong Lai Yin	NHG
Surgical Disciplines	Dr Edmund Chiong	NUHS

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