PROGRAMA DE INMERSIÓN EN LA INVESTIGACIÓN

TALLER DE ESCRITURA CIENTÍFICA

II PARTE ESCRIBO

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PhD. Biotecnología
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¿Qué es un artículo científico?

Documento publicado que describe **resultados originales de una investigación**

- Informa los resultados de una investigación científica, que responden a una hipótesis y que son comunicados por primera vez
- Los resultados publicados en el artículo científico deben ser reproducibles
- Debe comunicar de manera precisa, clara y breve los resultados, ideas y debates de su investigación
- Se escribe para otros, no para mí

Estructura general

I ntroduction	**¿Por qué?**
M ethods	**¿Cómo?**
R esults	**¿Qué?**
A nd	**y**
D iscussion	**¿Para qué?**

- Modelo que tiene por objetivo ayudar al autor a organizar la redacción del texto
- Su propósito es lograr una lectura fluida y clara

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Research Paper

Diabetic concentrations of metformin inhibit platelet-mediated ovarian cancer cell progression

Rafaela Erices[1,2], Sofía Cubillos[2], Raúl Aravena[2,3], Felice Santoro[2], Monica Marquez[2], Renan Orellana[1,15], Carolina Ramírez[2], Pamela González[2,4], Patricia Fuenzalida[2], María Loreto Bravo[2,4,5], Bárbara Oliva[2,4], Sumie Kato[1], Carolina Ibañez[5,6,7], Jorge Brañes[1], Erasmo Bravo[8], Catalina Alonso[8], Karen García[7,9], Clemente Arab[10], Vicente A. Torres[11,16], Alejandro S. Godoy[2,12], Jaime Pereira[6], Galdo Bustos[13], Julio Cesar Cardenas[13,14], Mauricio A. Cuello[1], Gareth I. Owen[2,4,5,7,16]

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Primera Página

- Título
- Nombre de todos los autores
- Departamentos y/o Instituciones de los autores (Afiliación)
- Nombre y correo de un autor para correspondencia (Investigador Académico Senior)

Research Paper

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Título

- Es la **etiqueta** que define el contenido del artículo
- Es lo que llevará a que la persona desee leerlo o no, por lo que debe ser **atractivo** para el público objetivo
- Debe ser conciso, sin abreviaturas y evitando comenzar con "estudio de…"

El tipo de título depende del estilo de la revista. Podemos encontrar:

- **Título Descriptivo**: Reseña el contenido de la investigación sin ofrecer resultados (Indica de qué se trata el estudio)

  Ejemplo: **Evaluation of azacitidine and entinostat as sensitization agents to cytotoxic chemotherapy in preclinical models of non-small cell lung cancer**

- **Título Informativo**: Comunica el resultado principal de la investigación (Indica el resultado del estudio)

  Ejemplo: Research Paper **Diabetic concentrations of metformin inhibit platelet-mediated ovarian cancer cell progression**

Autores

- Tienen la responsabilidad pública del contenido
- Deben anotarse de acuerdo al orden de importancia según su participación

- Su participación debe ser **sustancial** en:
  - Concepción, diseño, análisis
  - Preparación o crítica del manuscrito
  - Aprobación de la versión final

Nombre Bibliográfico Único

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- ***Apellidos compuestos:*** *todos unidos por guion Ruiz-de-la canal-castro, Sergio*

No es Autor

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- Aquel que sólo sugiere una idea genérica
- Aquel que recoge los datos sin interpretación de los mismos
- Aquel que hace el trabajo asistencial habitual
- Aquel que hace crítica puramente formal
- El jefe/autoridad (...por el hecho de serlo...)
- Para devolver favores
- Por acuerdo entre las partes

Resumen

- Debe informar al lector sobre el tema de la investigación que se presenta, indicando brevemente: Introducción, objetivos, metodología (según la revista), resultados, y conclusión
- Debe contener información específica de la investigación, que permita su hallazgo en una búsqueda bibliográfica

Algunos consejos para escribirlo:

- ✓ Utilizar tiempo pasado
- ✓ Que contenga las palabras claves del estudio
- ✓ Que el hallazgo de la investigación quede claro para el lector

Research Paper

Diabetic concentrations of metformin inhibit platelet-mediated ovarian cancer cell progression

ABSTRACT

Clinical studies have suggested a survival benefit in ovarian cancer patients with type 2 diabetes mellitus taking metformin, however the mechanism by which diabetic concentrations of metformin could deliver this effect is still poorly understood. Platelets not only represent an important reservoir of growth factors and angiogenic regulators, they are also known to participate in the tumor microenvironment implicated in tumor growth and dissemination. Herein, we investigated if diabetic concentrations of metformin could impinge upon the previously reported observation that platelet induces an increase in the tube forming capacity of endothelial cells (angiogenesis) and upon ovarian cancer cell aggressiveness. We demonstrate that metformin inhibits the increase in angiogenesis brought about by platelets in a mechanism that did not alter endothelial cell migration. In ovarian cancer cell lines and primary cultured cancer cells isolated from the ascitic fluid of ovarian cancer patients, we assessed the effect of combinations of platelets and metformin upon angiogenesis, migration, invasion and cancer sphere formation. The enhancement of each of these parameters by platelets was abrogated by the present of metformin in the vast majority of cancer cell cultures tested. Neither metformin nor platelets altered proliferation; however, metformin inhibited the increase in phosphorylation of focal adhesion kinase induced by platelets. We present the first evidence suggesting that concentrations of metformin present in diabetic patients may reduce the actions of platelets upon both endothelial cells and cancer cell survival and dissemination.

Introducción

Objetivo

Resultados

Conclusión

Palabras Clave

- Son las etiquetas del artículo
- Recogen el contenido de la investigación y sirven para recuperar fácilmente el documento en las bases de datos
- Para elegir palabras claves se pueden utilizar los siguientes buscadores: Medical Subject Headings (MESH)/ DeCS Health Sciences Descriptors
- De preferencia, algunas de estas palabras claves tienen que estar contenidas en el título

Palabras Clave

Ejemplo:

Research Paper

Diabetic concentrations of metformin inhibit platelet-mediated ovarian cancer cell progression

Células de cáncer de ovario

Keywords: *thrombocytosis, hemostasis, EA.hy926, SKOV3, UCI101*

Received: *July 27, 2016* ***Accepted:*** *January 27, 2017*

- ✓ Otras palabras claves que podrían haberse incluido: metformin, ovarian cancer

Introducción

- Describe el tema principal del estudio y las motivaciones para estudiarlo
- Se relaciona el estudio que se presenta con la literatura previa
- Señala la hipótesis y/u objetivos del estudio
- Se redacta en presente, pues hace referencia a reportes que son válidos en ese momento.

Research Paper

Diabetic concentrations of metformin inhibit platelet-mediated ovarian cancer cell progression

INTRODUCTION

Ovarian cancer is the fifth leading cause of cancer death in women and the most lethal gynecologic malignancy [1]. Thrombocytosis, high platelets counts, is observed in 10–57% of patients with cancer, which is prevalent in ovarian cancer, and been correlated with poor prognosis [1, 2]. Approximately one-third of women with recently diagnosed ovarian cancer have platelet counts exceeding 450,000/μL [3]. Furthermore, thrombocytosis is associated with several aggressive clinical features including, increased median preoperative serum CA-125 levels, advanced-stage disease and significantly decreased progression-free and overall survival (2.62 years compared with 4.65 years in those without thrombocytosis) [3]. Platelets has a fundamental role in hemostasis and coagulation [4]. Platelets are cytoplasmic fragments lacking a nucleus, which derive from the fragmentation of their precursor cells, megakaryocytes. As summarized in Figure 1, it has been widely reported that activated platelets can act directly upon the endothelium to promote angiogenesis and act upon the cancer cell to contributes to tumor-promoted angiogenesis, tumor-development and metastasis [1, 5]. Platelet activation can occur upon the binding of cell surface agonists such as thrombin [6] or the removal of platelets from their environment, which contains activation inhibitors such as prostaglandins [2, 7]. Platelet activation leads to aggregation, exposure of membrane proteins and release the content of their granules, which in the case of tumor cells is implicated in the promotion of angiogenesis, growth, survival and metastasis [1, 5, 8]. In *in vitro* and *in vivo* murine models it has been shown that platelets participate in cancer development and protect tumor cells in circulation from elimination by the immune system [1]. In a previous work, we showed that platelets could act as chemoattractants to cancer cells, increase the expression of metastasis initiating cell markers and enhance cancer sphere formation (Figure 1) [2]. These influences may enable tumor cells to arrest in the vasculature, mediate an inflammatory response produced by the interaction of platelets with the tumor microenvironment and thus favor proliferation and angiogenesis [1, 9].

Metformin is widely used to treat type 2 diabetes and pre-diabetic syndromes modulating glucose metabolism and fatty acids. Its primary action is to inhibit hepatic glucose production, but it also increases the sensitivity of peripheral tissues to insulin [10]. To date, several epidemiological studies indicate that the use of metformin in patients with cancer would be beneficial, especially observed in an increase in disease-free survival [11, 12]. These studies initiated the scientific interest in determining the mechanism of action by which metformin delivers anti-cancer benefits. Habitual clinical dosing regimens of metformin hydrochloride tablets generally result in steady state plasma concentrations of less than 1 mg/mL, which are achieved within 24 to 48 hours (U.S. Food & Drug Administration). During controlled clinical studies of metformin, maximum plasma metformin levels do not exceed 5 mg/mL (30 μmol/L). Furthermore, it has been reported that the maximum plasmatic concentration in diabetic patients is within a range of 1-4 mg/ml, which corresponds to 6–24 μM respectively (U.S. Food & Drug Administration). Furthermore, Lalau and colleagues showed that the mean + standard deviation plasma concentrations were 2.7 ± 7.3 mg/L (16 ± 44 μmol/L) in a total of 467 patients [13]. However, the concentrations used in most published *in vitro* and *in vivo* studies are several times higher than maximum plasma concentrations that would be achieved with the doses of metformin used by diabetic patients [14]. Thus, the currently published mechanism of action may help promote use of high dose of metformin as a stand-alone cancer treatment, however these mechanisms may not necessarily explain why diabetic concentrations have beneficial effects on cancer incidence and survival [15, 16].

Currently, few studies are available regarding the effects that would have metformin on platelet function. Several studies indicate that in patients with type 2 diabetes mellitus, metformin would be beneficial in maintaining hemostasis in these patients [17]. Several years ago, the effect of platelet function in the presence of metformin was determined, in response to different stimuli, including adrenaline and ADP. Authors observed that the presence of metformin decreases platelet function (aggregation) in response only to the combined stimulus of ADP and adrenaline, but not against simple stimuli [18]. A recent trial [19] has documented that metformin decreased mean platelet volume (MPV), which is known to be increased in diabetes mellitus and has been correlated with vascular complications [20].

Our published results have shown that the use of metformin in concentrations approved for use in diabetics (micromolar range) has no effect on cell proliferation, but can allow ovarian cancer cells to overcome resistance to carboplatin [21]. Furthermore, in a previous publication we demonstrated that platelets could promote cell migration, EMT and sphere formation in cultures of ovarian cancer [2]. Given the increasing literature suggesting that metformin could have beneficial effects upon ovarian cancer patients and that metformin can modify platelet function, we speculate that the use of micromolar concentrations of metformin alone would not have significant effects on angiogenesis, migration and epithelial-mesenchymal transition, but may abrogate platelet-mediated progression of ovarian cancer.

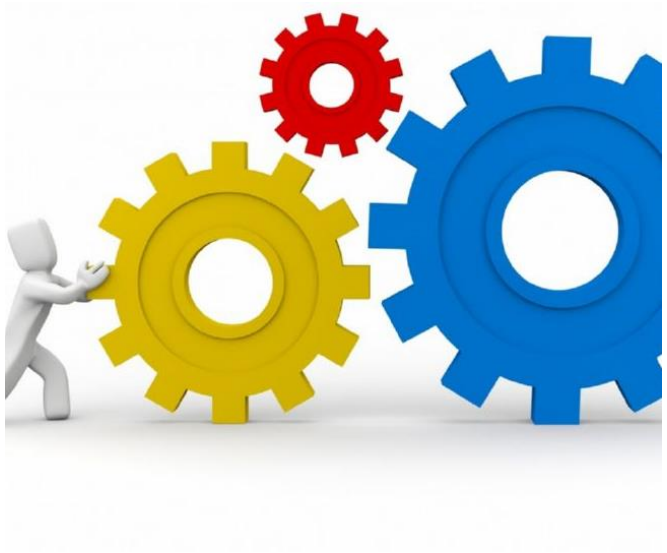
Motivación para realizar la investigación y que llevaron a la hipótesis

Tema y literatura previa

Motivación para realizar la investigación

Metodología/ Materiales y Métodos

- Describe específicamente el diseño experimental para realizar los objetivos y resolver la hipótesis del estudio
- En esta sección se suelen enunciar los materiales utilizados, como equipamiento, reactivos, software, etc., indicando las marcas de tales materiales
- No especifica marcas comerciales ni modelos específicos si varios equipos pueden hacer lo mismo (Ej. Computador hp Windows 10)
- ✓ Escribir en pasado, pues representan acciones ya realizadas
- ✓ Usar nombres genéricos para los compuestos químicos si no hay diferencias importantes entre las marcas (Ej. Paclitaxel, Paracetamol, etc.)



Diabetic concentrations of metformin inhibit platelet-mediated ovarian cancer cell progression

Materiales

MATERIALS AND METHODS

Cell line culture

The ovarian cancer cell lines SKOV3 and UCI101 were maintained in Dulbecco's modified Eagle medium (DMEM)/F12 supplemented with 10% fetal bovine serum (Invitrogen, Carlsbad, California, USA). HUVEC were isolated from umbilical cords obtained with patient consents and approved by the ethical committee at the Hospital Clínico Universidad Católica de Chile. HUVEC were obtained by collagenase treatment and maintained in Human Endothelial SFM medium (Life Technologies, Grand Island, NY, USA) supplemented with 10% fetal bovine serum (FBS) and endothelial cell growth supplement (6 mg/ml final concentration) (Merck Millipore, Billerica, MA, USA). These cells were used until passage five. Endothelial EA.hy926 cells were maintained in Iscove's Modified Dulbecco's Media (IMDM) supplemented with 10% fetal bovine serum. For all experiments, endothelial cells were used until passage six. Metformin was solubilized in water (D150959; Sigma-Aldrich, St. Louis, MO, USA), and Compound C (Dorsomorphin) was solubilized in DMSO (ab120843; Abcam, Cambridge, UK).

Primary cell culture of ovarian cancer

All ovarian cancer samples used in primary cultures were obtained from patients with high-grade ovarian carcinomas, with signed informed consent and with institutional ethical committee approval from the Pontificia Universidad Católica de Chile and all of the participating Chilean hospitals. The hospitals included the Cancer Center at the Pontificia Universidad Católica, Hospital Gustavo Fricke, Hospital Sótero del Río, and Hospital Luis Tisné. For all the experiments, primary cultures were used until passage two.

previously described by Orellana et al., 2015 [2]. Briefly, venous blood was collected from healthy volunteers (not taking anti-platelet drugs). Platelet-containing medium was added to the culture dish (final concentration 1.5×10^5 platelets/μL) containing already seeded cell lines SKOV3, UCI101, EA.hy926, HUVEC, and the human ascites primary culture. After 12 hours or 24 hours of incubation, the cell monolayer was washed three times with PBS to eliminate the platelets in suspension.

Tube formation assay (angiogenesis *in vitro*)

The tube formation assay was performed using EA.hy926 and HUVEC cells as described [55]. Briefly, EA.hy926 cells or HUVEC cells (30,000 cells/well, in DMEM/F12 0% FBS) were plated on top of matrigel coated plates (48 well plates) and incubated with the treatments at 37°C for 12 h. In the case of the treatments of conditioned media from ovarian cancer cell lines SKOV3 and UCI101, these cells were treated for 24 h at 37°C in the presence of either platelets, metformin or both. After 24 h, the conditioned media was removed and centrifuged at 2700 g for 9 min at 4°C to remove cell or platelet debris. This conditioned media was used to resuspend endothelial cells before seeding onto matrigel. Cultures were photographed using 4× and 10× of magnification and all results were quantified using the plugin "Angiogenesis Analyzer" from image J v1.6. For angiogenesis score we used 4X images using the following formula:

Angiogenic score = number of branches × total branch length

Scratch assay

SKOV3, UCI101 and EA.hy926 cells were cultured in 6 well plates until 100% confluence, and then a vertical and horizontal wound (scratch) was introduced through the cell monolayer using a fine pipette tip. The culture medium was replaced with fresh DMEM/F12 containing 5% charcoal treated serum in the presence or absence of platelets (1.5×10^5 platelets/μL) and or metformin (20 μM). In the case of EA.hy926, the culture medium was replaced with fresh DMEM/F12 in the absence of FBS. Wound closure was assessed by photography at 5, 10 and 24 hours, and quantified using the Infinity Analyze v6.3 (Lumenera Corporation, Ontario, Canada).

Cell cycle analysis

Flow cytometry (FACSsan, Beckton Dickinson, Franklin Lakes, New Jersey, USA) was performed at Clínica Tabancura, Chile. Briefly, cells were collected and washed 2 times with cold phosphate-buffered saline.

Metodología y Materiales

Resultados

- Son las respuestas a la hipótesis planteada en la investigación, sirven para dar a conocer los hallazgos respecto a un tema de estudio.
- Se presentan en formato de texto, tablas, gráficos y figuras
- Los resultados se escriben en pasado, pues fueron encontrados mucho antes de escribir el artículo

Hipótesis

ovarian cancer [2]. Given the increasing literature suggesting that metformin could have beneficial effects upon ovarian cancer patients and that metformin can modify platelet function, we speculate that the use of micromolar concentrations of metformin alone would not have significant effects on angiogenesis, migration and epithelial-mesenchymal transition, but may abrogate platelet-mediated progression of ovarian cancer.

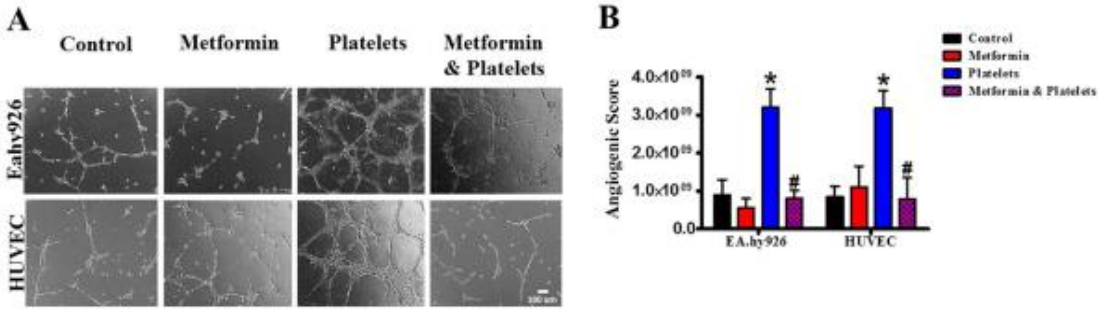
Concentraciones micromolares (Diabéticas) de Metformina inhiben el efecto de las plaquetas sobre la angiogénesis, migración y transición Epitelio-Mesénquima

Research Paper

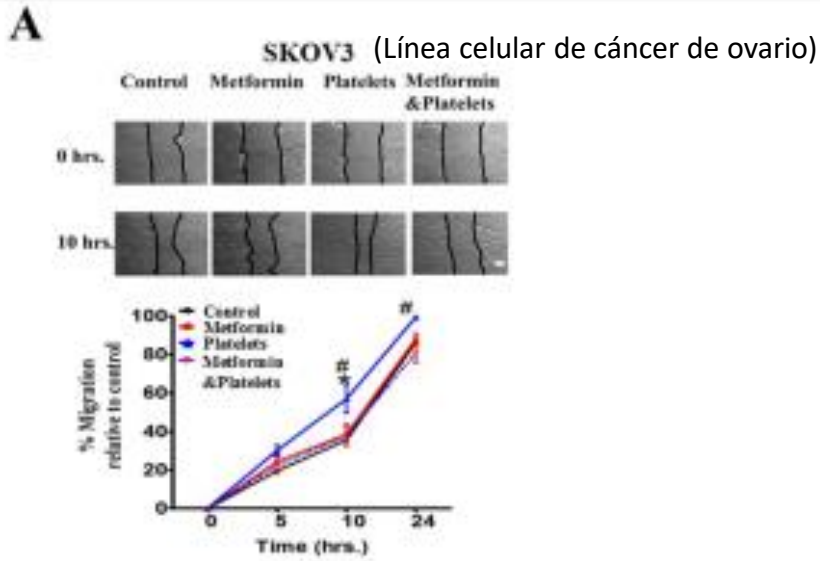
Diabetic concentrations of metformin inhibit platelet-mediated ovarian cancer cell progression

Resultados de acuerdo a la hipótesis

Metformina evita que las plaquetas promuevan la angiogénesis

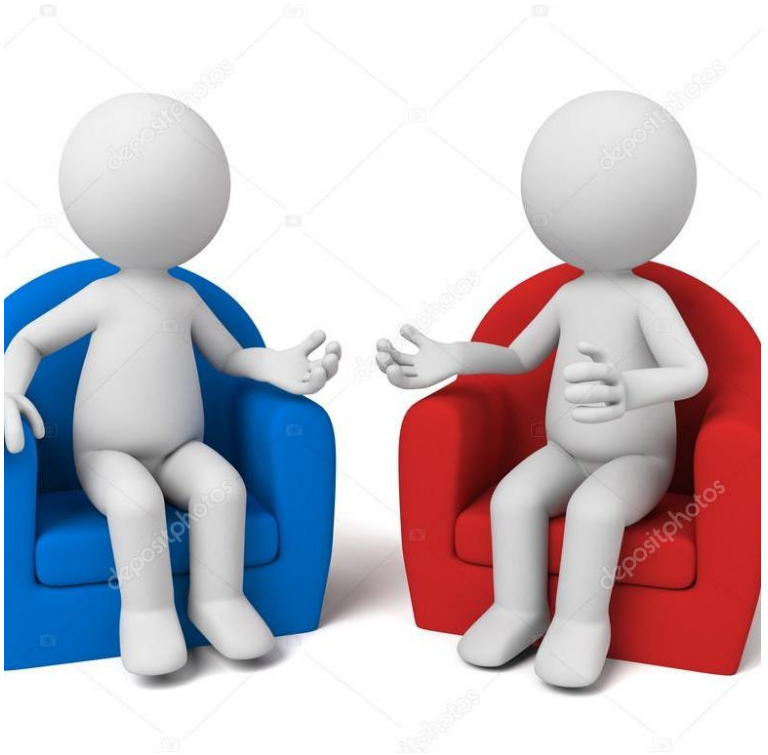


Metformina evita que las plaquetas promuevan la migración en células de cáncer de ovario



Discusión

- Se interpretan los resultados obtenidos y se analizan respecto a resultados similares de otras investigaciones
- Comenta los descubrimientos inesperados
- En ocasiones Discusiones y Conclusiones van en la misma sección (no confundir)
- Al debatir y opinar sobre contenidos de otros autores se escribe en presente, pues son conocimientos actuales que se usan como referencia. En cambio, cuando se comentan los resultados obtenidos se escribe en pasado.



Research Paper

Diabetic concentrations of metformin inhibit platelet-mediated ovarian cancer cell progression

DISCUSSION

Metformin is an anti-diabetic drug, widely used for first-line treatment of type 2 diabetes mellitus, that has received a lot of attention from the oncology

Breve introducción

The platelet-cancer cell interaction and metformin: angiogenesis

The association between platelets and the process of tumor angiogenesis has been recognized [38]. The role of platelets in the regulation of angiogenesis *in vivo* was first documented in a cornea angiogenesis model, in which platelets were shown to support angiogenesis and prevent leakage, and hemorrhage from the newly formed vessels [32]. The participation of platelets in angiogenesis has been observed *in vitro* with the platelets promoting the formation of capillary structures in Matrigel assays [39].

As a first line treatment in diabetes metformin has demonstrated improved peripheral insulin sensitivity and glucose transportation after treatment in diabetic patients [40]. However, other studies have shown that metformin exerts other effects beyond those on glucose metabolism, as by example to modulate inflammation in polycystic ovary syndrome [41] and to exert antiproliferative actions specially in cancer cells [42, 43]. These observations have expanded the range of actions of this drug that may explain several improvements not associated with its anti-hyperglycemic action [28]. Xavier et al., [28] studied the effects of systemic treatment of metformin in the sponge model to evaluate its actions on early steps of the formation of the fribrovascular tissue. They found that metformin has a regulatory function on components of inflammatory angiogenesis, attenuating vascularization.

Resultados de otros estudios relacionados

In the present study, we observed that the addition of platelets to ovarian cancer cells brought about an increase in the balance of pro-angiogenic factors, as demonstrated by the formation of capillary-like structures upon co-incubation of the resulting conditioned medium (removed at 24 hours) with endothelial cells on matrigel. The coincubation of platelets in solely culture medium for the same period before incubation with endothelial cells did not result in changes in angiogenesis, most likely due to the short half-life of the growth factors released by platelets. Interestingly, the presence of metformin reduced this angiogenic potential in both cell lines tested and three of the four cultures of cancer cells isolated from the ascites of high-grade serous papillary ovarian cancer. In one culture, metformin did not reduce platelet-increased angiogenesis. While in the sphere forming assays and the migration assay we clearly see an effect of metformin upon the cancer cell (inhibiting the platelet-effect of the latter two processes) we cannot fully rule out the possibility that Metformin remains in the conditioned medium and thus inhibits angiogenesis by a direct effect upon endothelial cells. However, an anti-angiogenic effect

Resultados del presente estudio

Conclusiones

- Explica brevemente los resultados más relevantes
- Resalta los hallazgos en el estudio de un tema, y su aporte al conocimiento en el campo
- Indica los vacíos o limitaciones que presenta la investigación, los cuales pueden ser resueltos por otro(a) investigador(a)
- Entrega recomendaciones para estudios posteriores
- Comenta las perspectivas futuras de la investigación

Research Paper

Diabetic concentrations of metformin inhibit platelet-mediated ovarian cancer cell progression

CONCLUSIONS

Breve introducción

The published literature is now showing an irrefutable distinction between patient-approved pharmaceutical and super pharmaceutical doses of metformin in the field of oncology. These observations reflect the diversity of action of metformin, further emphasizing that there are still many mechanisms of action upon the cancer cell that need to be identified. This study adds further argument that metformin research should be separated into two disciplines. Firstly, the use of concentrations below 50 micromolar would help elucidate how metformin lowers the cancer incidence and improves outcome in patients taking the drug. Secondly, the use of higher (millimolar) concentrations will evaluate if metformin alone, or when incorporated into future cancer therapeutic regimes, could be beneficial to patient outcome.

Recomendaciones para estudios posteriores

Resultados y aporte

The results presented in this paper show a protective action of metformin upon platelet-mediation processes involved in cancer progression. This may help us to understand the beneficial clinical effects shown by this drug in patients with ovarian cancer. This anti-platelet action of metformin may not just be limited to cancer. Metformin use has been demonstrated to reduce mean platelet mass and volume in patients at risk for atherosclerotic disease [19]. Thus, in conjunction with positive effects on vascular adhesion, cholesterol levels and inflammatory markers, metformin through the reduction of platelet volume and action may have a role in reducing the development of atherosclerosis. Future investigation will elucidate whether the action of metformin on platelet activity is also relevant in insulin resistance and polycystic ovary syndrome. Herein, we demonstrate that metformin possesses actions that further support the ongoing clinical trials evaluating the inclusion of metformin alongside conventional cancer therapy.

Perspectivas futuras

Agradecimientos

- Es el reconocimiento a las personas que ayudaron en la investigación
- Personas que asesoraron
- Los apoyos financieros
- Revisores
- Proveedores de material
- Servicios

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PMCID: PMC3141650
PMID: 21729302

Bradykinin promotes migration and invasion of human immortalized trophoblasts

Rafaela Erices,[1] Jenny Corthorn,[1,2] Francisco Lisboa,[1] and Gloria Valdés[✉1,2]

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Referencias Bibliográficas

- Citar siguiendo las indicaciones de "Guide for authors/Instructions for authors" que presentan las revistas
- Permite comprobar las fuentes bibliográficas

- ✓ *Citar referencias que provengan de la revista de interés*
- ✓ *Evitar exceso de auto-citas (para evitar manipular los factores de impacto)*

Resumiendo:

- **Título:** Etiqueta que define el contenido del artículo, por lo que debe ser preciso y conciso
- **Autores:** Son los responsables del estudio, su participación debe ser sustancial (concepto, diseño, análisis y preparación y aprobación de la versión final)
- **Resumen:** Informa brevemente al lector el tema de estudio (considerando Introducción, Objetivos, Metodología, Resultados y Conclusión)
- **Introducción:** Describe el tema principal del estudio y las causas para estudiarlo

Resumiendo:

- **Metodología**: Describe específicamente la metodología experimental para resolver la hipótesis del estudio
- **Resultados:** Se presentan como texto, tablas, gráficos y/o figuras. Responden a la hipótesis y objetivos de la investigación
- **Discusión:** Interpretación de los resultados obtenidos, contrastados con resultados de estudios similares previos
- **Conclusión:** Explica brevemente los resultados más relevantes obtenidos, su aporte al conocimiento, sus limitaciones, y perspectivas futuras

Resumiendo:

- ✓ *Evitar el uso de términos ambiguos, tales como: frecuentemente, regularmente y periódicamente*
- ✓ *Para que la investigación pueda ser reproducible, el lector necesita entender exactamente qué se hizo , cuándo y cómo*
- ✓ *Seleccionar un Nombre bibliográfico único, ya que esa será la forma en que el autor será encontrado siempre en las bases de datos*
- ✓ *Los resultados del estudio y su impacto dentro de su área de investigación deben ser claros y precisos*

Revistas:

Revistas científicas multidisciplinarias de prestigio

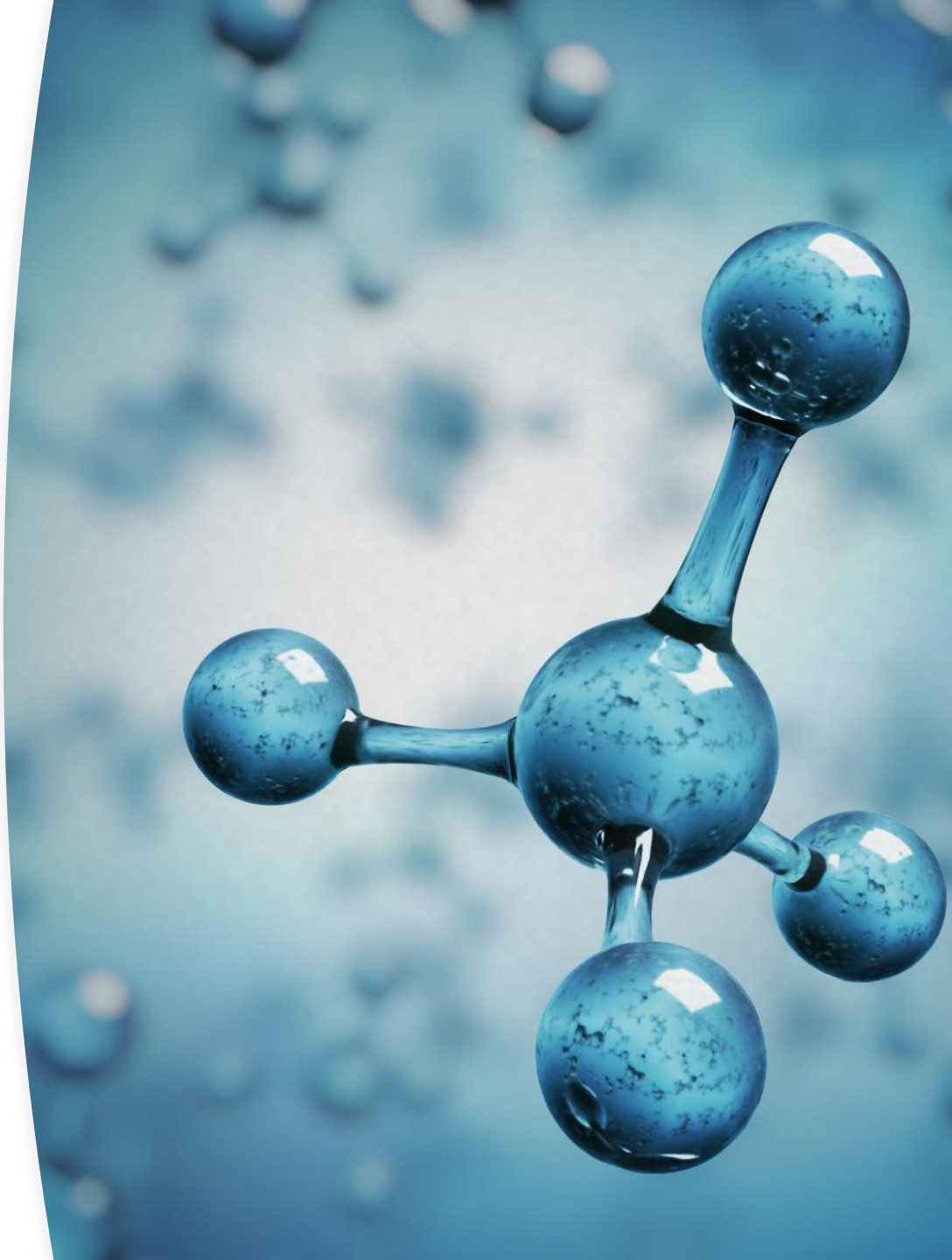
Journal	Impact factor (2017)
Nature	41.577
Nature Communications	12.353
Philosophical Transactions of the Royal Society A	2.746
PLOS ONE	2.766
Proceedings of the National Academy of Sciences	9.504
Proceedings of the Royal Society A	2.410
Science	37.205 (2016 year data)
Science Advances	11.51
Scientific Reports	4.122

Revistas científicas específicas de prestigio

- **Algunos ejemplos en Biología:**
  - BioEssays
  - Biological Reviews
  - Biophysical Journal
  - Cell
  - eLife
  - International Journal of Biological Sciences
  - Journal of Cell Biology
  - Journal of Molecular Biology

Revistas científicas específicas en crecimiento (algunos ejemplos)

- International Journal of Architectonic, Spatial, and Environmental Design
- Molecular Microbiology
- Oral Diseases
- Revista Chilena de Pediatría
- IAWA Journal
- Revista Brasileira de Ciências do Esporte
- Revista de Derecho [Chile]
- Revista de Estudios Histórico Jurídicos
- Revista Médica de Chile

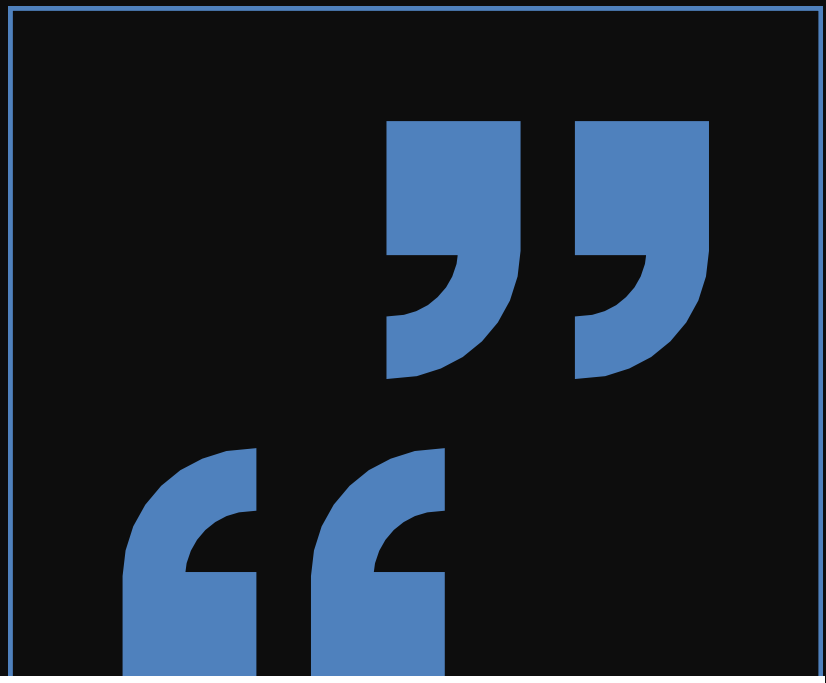


Ética en la publicación

- Normas que garantizan la calidad y confiabilidad de las publicaciones científicas. Aseguran al lector que la información disponible sea fidedigna, y que las personas que hicieron la investigación reciban crédito por sus ideas.

AUTENTICIDAD Y PRECISIÓN

- Proporcionar datos completos (no solo que apoyen la hipótesis)
- Evitar la manipulación inadecuada de imágenes
- Usar análisis estadístico apropiado



Plagio

- Copiar el trabajo de otro: copia textual, gráfica o de ilustración, sin citar mediante una referencia bibliográfica explícita

Algunos casos que se pueden encontrar:

- Parafraseo: tomar párrafos de otros autores, haciendo intercambio del orden de frases y reemplazando palabras por sus sinónimos
- Copia sustancial: pero con maquillaje más profundo que el parafraseo
- Autoplagio: cuando el autor se copia a sí mismo
  - ✓ *Cuando el artículo publicado deriva de una tesis o trabajo de grado (que se publica al interior de una Universidad) no corresponde a un autoplagio*
- Cuando se cita explícitamente al otro autor, pero no se delimita adecuadamente la copia literal del texto

Fraude

Manipulación de los datos con el fin de ajustarlos para que soporten las conclusiones del estudio.

Retractación:

- Si un autor descubre un error significativo o inexactitud en su propio trabajo publicado, es su obligación notificar de inmediato a la editorial de la revista para retractarse o corregir el artículo lo antes posible

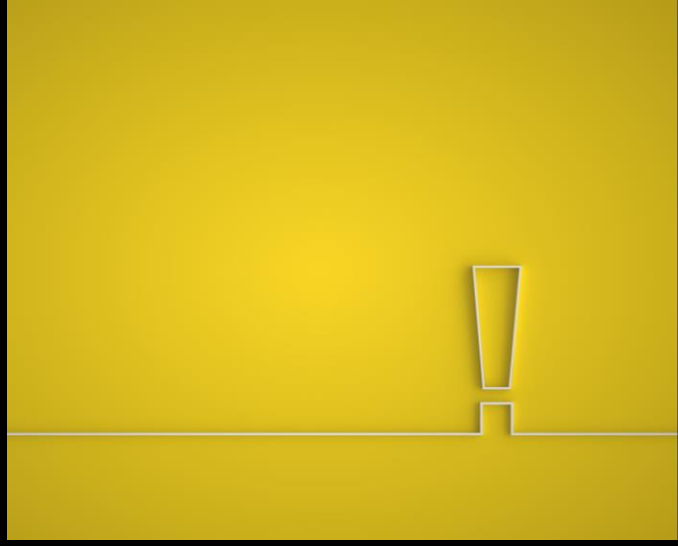
- Si un editor descubre que una obra publicada contiene un error importante, es obligación del(la) autor(a) retractarse a la mayor brevedad posible, corregir el artículo o aportar pruebas al(la) editor(a) de la corrección realizada al documento original

SANCIONES

- Si el (la) editor(a) de una revista confirma una publicación redundante, deberá informar a todas las partes involucradas, editores de las revistas involucradas así como a la institución a la que pertenece el(la) infractor(a)

- Adicionalmente a las sanciones que pudiera dar la institución a la que pertenece el(la) infractor(a), una revista podría vetar al(la) autor(a) y rechazar todos los manuscritos en revisión y listos para su publicación

- Para más detalle sobre estos procedimientos se puede referir al Comité de ética de la publicación (COPE, por su sigla en inglés) en el sitio web http://publicationethics.org.



Referencias

Algunas claves para escribir correctamente un artículo científico. Villagrán y cols. 2009

Escribir y publicar un artículo científico original. Rafael Ferriols y Francisco Ferriols, 2005.

Si tu intención es describir la verdad, hazlo con sencillez y la elegancia déjasela al sastre.

Albert Einstein (1879-1955)

¡¡Gracias por su atención!!

CONTACTO

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