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Artículo de investigación

Exposición elevada a plomo en mujeres embarazadas de una comunidad alfarera en México: descripción de fuentes y papel del consumo de calcio.

Elevated Lead Exposure in Pregnant Women from a Pottery-Making Community in Mexico: Description of Sources and the Role of Calcium Intake

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Resumen

Antecedentes: la exposición a plomo (Pb) es preocupante en mujeres embarazadas de comunidades alfareras.
Objetivo: evaluar la exposición a Pb en mujeres embarazadas de una comunidad alfarera, mediante biomonitoreo de sangre capilar, su correlación con fuentes domésticas y de producción de LBV, así como su consumo de calcio (Ca).
Metodología: estudio descriptivo de ocho mujeres embarazadas expuestas a Pb. Se determinó Pb en sangre capilar, suelos y piezas de LBV, así como información socioeconómica, clínica/nutricional y consumo de Ca.
Resultados: todas las mujeres presentaron Pb en sangre elevado. Se observó una correlación positiva y significativa entre Pb en sangre y Pb en suelo doméstico. El 50% tuvieron una ingesta de Ca insuficiente con concentraciones altas de Pb.
Conclusiones: las mujeres embarazadas de esta comunidad alfarera presentaron exposición elevada a Pb relacionada al entorno doméstico y productivo. La baja ingesta de calcio podría contribuir a una mayor carga corporal de Pb.

Palabras clave: intoxicación por Pb, embarazo, consumo de Ca, México.

Abstract

Background: *Lead (Pb) exposure is a concern for pregnant women in pottery communities.*
Aim: *To assess Pb exposure in pregnant women in a pottery-making community through capillary blood biomonitoring, its correlation with domestic and LBV production sources, and calcium (Ca) consumption.*
Methodology: *Descriptive study of eight pregnant women exposed to Pb. Pb was determined in capillary blood, soil, and LBV pieces, as well as socioeconomic, clinical/nutritional information, and Ca consumption.*
Results: *All women had elevated blood Pb levels. A positive and significant correlation was observed between blood Pb and domestic soil Pb. Fifty percent had insufficient Ca intake with high Pb concentrations.*
Conclusions: *Pregnant women in this pottery-making community had high exposure to Pb related to their domestic and productive environments. Low calcium intake could contribute to a higher body burden of Pb.*

Keywords: *Pb poisoning, pregnancy, Ca consumption, Mexico*

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INTRODUCCIÓN

El plomo (Pb) es un metal pesado que se encuentra de forma natural en el medio ambiente, especialmente en suelos volcánicos.[1] Sin embargo, debido a las actividades humanas, se ha extendido a productos de consumo comunes, como pinturas, casas antiguas, cosméticos y juguetes.[2-4] Las principales vías de exposición al Pb incluyen la vía digestiva a través del consumo de alimentos y agua contaminados, así como por la inhalación.[5] Para los seres humanos, no se conoce ningún umbral seguro; incluso dosis mínimas pueden ser tóxicas para el organismo.[2,5,6] De acuerdo con la Organización Mundial de la Salud (OMS), el Pb se encuentra entre las 10 sustancias más peligrosas y preocupantes a nivel mundial.[7]

Actualmente, la principal fuente de exposición a Pb entre la población general de México es el uso de loza de barro vidriada con Pb (LBV) para la preparación, almacenamiento y consumo de alimentos.[8] Estas piezas están recubiertas con esmalte que contiene óxido de Pb (PbO), también conocido como "greta", que puede filtrarse en los alimentos y bebidas.[9-11] Los alfareros que producen LBV representan un grupo especialmente vulnerable, ya que están directamente expuestos al PbO al esmaltar piezas de cerámica; mientras que los miembros de sus familias también comparten una exposición para-ocupacional, ya que los talleres y los hornos suelen estar ubicados dentro de sus hogares.[12]

En México, la Norma Oficial Mexicana NOM-199-SSA1-2000 establece el valor de referencia de $\geq$ 5 µg/dL para intoxicación por Pb en niños y mujeres embarazadas o en periodo de lactancia.[13] Sin embargo, la NOM-047-SSA1-2011 establece un valor de referencia de 10 µg/dL para las mujeres en edad reproductiva ocupacionalmente expuestas,[14] sin considerar la etapa fisiológica en la que se encuentre la mujer.

Estudios previos en población de mujeres alfareras han reportado concentraciones elevadas de Pb en sangre, con valores que oscilan entre 5.8 µg/dL y 65 µg/dL, observándose las concentraciones más altas en aquellas cuyos hornos se encontraban situados en el interior o en las inmediaciones del hogar.[10,12] Esta situación es particularmente preocupante en mujeres durante la etapa del embarazo, ya que el Pb tiene la capacidad de atravesar la barrera fetoplacentaria, afectando todos los órganos y sistemas del feto en desarrollo.[15]

Además de la exposición ambiental directa, las mujeres expuestas a Pb durante períodos prolongados pueden acumularlo en el tejido óseo.[16] Durante el embarazo, el aumento en el recambio óseo, necesario para satisfacer los requerimientos fetales de calcio (Ca), favorece la movilización del Pb almacenado del hueso al torrente sanguíneo materno, convirtiendo a la madre en una fuente endógena adicional de exposición fetal.[17] Este proceso se ha asociado con un mayor riesgo de efectos adversos durante el embarazo como preeclampsia,[18] bajo peso al nacer[19] e incremento de la mortalidad infantil,[20] así como efectos a largo plazo en la descendencia, induciendo neurotoxicidad mediante la alteración de procesos dependientes del Ca, interfiriendo con la señalización celular, la neurotransmisión y la diferenciación neuronal,[21] provocando un daño neurológico irreversible, que compromete la capacidad cognitiva y el comportamiento.[22,23]

Evidencia experimental y epidemiológica indica que la deficiencia de nutrimentos inorgánicos como Ca, hierro (Fe) y zinc (Zn) incrementa la absorción intestinal y la toxicidad de metales pesados como el Pb, debido a la competencia con las proteínas transportadoras involucradas en la absorción intestinal de metales divalentes.[24,25] Además, estudios previos en mujeres embarazadas mexicanas han mostrado que la suplementación con Ca puede reducir la absorción de Pb.[26]

En México, el consumo insuficiente de nutrimentos inorgánicos continúa siendo un problema de salud pública relevante. De acuerdo con datos de la Encuesta Nacional de Salud y Nutrición (ENSANUT) 2016, una proporción considerable de la población mexicana no alcanza las recomendaciones dietéticas para diversos nutrimentos inorgánicos, entre ellos el Ca.[27] Esto evidencia brechas persistentes en la calidad de la dieta a nivel nacional, así como un posible aumento en la absorción y toxicidad de metales pesados como el Pb. Sin embargo, hasta el momento no existe evidencia sobre concentraciones de Pb en mujeres embarazadas expuestas a este metal tóxico a través de la alfarería de manera ocupacional y/o para-ocupacional. Por lo tanto, el objetivo de este estudio fue evaluar la exposición a Pb en mujeres embarazadas residentes de una comunidad alfarera en Puebla, México, mediante biomonitoreo de sangre capilar e investigar su correlación con fuentes relacionadas a la producción de LBV, así como explorar el papel del consumo de Ca en dicha exposición.

METODOLOGÍA

POBLACIÓN DE ESTUDIO

Se realizó un estudio descriptivo en la comunidad alfarera ubicada en el municipio de Cohuecan, Puebla, México. Entre agosto de 2024 y abril de 2025, a través del centro de atención primaria de salud de la comunidad (Unidad de Medicina Rural UMR Cohuecan zona 14), se invitó a participar a mujeres embarazadas que llevaran su seguimiento de atención prenatal en este centro de salud, mayores de 18 años, con embarazo confirmado de 20 semanas o más de edad gestacional (EG); y que tuvieran una exposición al Pb de manera ocupacional (trabajan

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directamente en talleres de alfarería) y/o para-ocupacional (algún miembro de la familia que vive en la misma casa trabaja en la alfarería y/o el taller de alfarería se encuentra en el mismo lugar donde habita la mujer embarazada). Las mujeres que aceptaron participar firmaron una carta de consentimiento informado. Una vez que las mujeres aceptaron participar, se agendaron dos visitas, la primera en el centro de salud, en la cual se realizaron las determinaciones de Pb en sangre capilar y la recolección de información sociodemográfica y clínico-dietética. La segunda visita se realizó en los hogares de las participantes para determinar las concentraciones de Pb en suelos de los hogares, talleres y las piezas producidas en sus talleres. Este estudio fue revisado y aprobado por el comité de ética de la Universidad Iberoamericana de la Ciudad de México con el número CEI: 15/24.

DETERMINACIONES DE PB

CUANTIFICACIÓN DE PB EN SANGRE CAPILAR

Durante la primera visita la cuantificación de la concentración de Pb en sangre capilar de las participantes se realizó por personal previamente capacitado y estandarizado. A cada participante se le tomaron mediciones por duplicado y se promediaron. Cada muestra consistió en 50 µL de sangre capilar, los cuales fueron analizados en un equipo de voltamperometría de disolución anódica con un equipo portátil LeadCare II (Magellan Diagnostics, North Billerica, MA, EE. UU.) con un rango de detección de 3.3 a 65 µg/dL.[28]

CUANTIFICACIÓN DE PB EN SUPERFICIES DOMÉSTICAS

En la segunda visita, se realizó la cuantificación de Pb en suelos de los hogares de las participantes. Las mediciones se realizaron en tres tipos de muestras: 1) el suelo de las habitaciones y zonas verdes; 2) el suelo del taller y 3) las piezas fabricadas en el taller. Un mínimo de una medición fue registrada para cada tipo de muestra de suelo y pieza. Las muestras fueron analizadas a través de un equipo portátil de espectrofotometría de fluorescencia de rayos X (XRF) (*Thermo Fisher Scientific* modelo NITON XL3T, Waltham, MA, EE. UU.). El equipo se configuró previamente en el modo "Medio ambiente y suelo" para el suelo de las habitaciones y el taller, y en el modo "Cerámica" para las piezas. El rango de detección de este equipo es de 5-10 ppm.[29]

ESTIMACIÓN DE LA INGESTA DE CA

Para la estimación de la ingesta de Ca se aplicó un cuestionario de frecuencia de consumo de alimentos semicuantitativo validado (CFCA).[30] Este instrumento fue diseñado para recopilar información sobre la dieta consumida durante los siete días previos a la aplicación del cuestionario y fue administrado por un entrevistador capacitado. El cuestionario recaba información sobre 102 alimentos, agrupados en 14 categorías: productos lácteos, frutas, verduras, comida rápida casera, carnes, embutidos, huevos, pescado y marisco, legumbres, cereales y tubérculos, productos de maíz, bebidas, aperitivos, dulces y postres, sopas, cremas y pastas, salsas, limón y tortillas. Para cada alimento, se estableció un tamaño de porción estándar y nueve opciones de frecuencia de consumo que van desde "nunca" hasta "seis o más veces al día". Como ayudas visuales para estimar el tamaño de la porción real se utilizaron cucharas y una taza estandarizada. Estas estimaciones se compararon con las tablas de composición de alimentos de la Base de Alimentos de México (BAM) y los valores de frecuencia se clasificaron y se multiplicaron por la cantidad de Ca de cada alimento; las fuentes alimentarias y el consumo de suplementos fueron sumados para cuantificar la ingesta total de Ca por día (mg/d).

OTRAS VARIABLES DE ESTUDIO

Adicionalmente, se administraron cuestionarios a las participantes para recopilar información sociodemográfica, socioeconómica y clínico-dietética. Para estimar el nivel socioeconómico se incluyó el cuestionario de la Asociación Mexicana de Agencias de Inteligencia de Mercado y Opinión (AMAI 2022),[31] que va desde el nivel A/B (nivel más alto) hasta el E (nivel más bajo). Además, se utilizó el cuestionario de la Escala Latinoamericana y del Caribe de Seguridad Alimentaria (ELCSA),[32] que evalúa las condiciones de inseguridad alimentaria clasificándose en seguridad, inseguridad leve, moderada y grave. También se aplicó un historial médico y ginecológico-obstétrico. Respecto a las medidas antropométricas, fueron obtenidas por personal capacitado y estandarizado, además, se obtuvieron por duplicado y se promediaron. El peso se obtuvo a través de una báscula (modelo SECA 813, Hamburgo, Alemania) con precisión de ± 0.1 kg.[33] Las mediciones de la presión arterial se tomaron de acuerdo con las normas establecidas por la Organización Panamericana de la Salud (OPS),[34] utilizando un esfigmomanómetro digital (OMRON, modelo HEM-7120, Kioto, Japón), con una precisión de ± 3.0 mmHg.[35] La estatura fue obtenida de los registros de enfermería. Finalmente, el índice de masa corporal (IMC) se calculó dividiendo el peso (kg) por la estatura (m) al cuadrado (kg/m^2).

ANÁLISIS ESTADÍSTICO

La descripción de las características sociodemográficas, socioeconómicas y clínico-dietéticas fue presentada como frecuencias y porcentajes para las variables categóricas y como medias y desviación estándar (DE) para las variables continuas. Para las concentraciones de Pb en sangre

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capilar, suelo de los hogares, suelo en los talleres y en piezas elaboradas en los talleres, estas fueron presentadas como medias y DE y para el total de las mediciones dada la naturaleza de la distribución se presentaron como medianas y rango intercuartílico. Asimismo, las concentraciones de Pb en sangre capilar se compararon con los valores de referencia de la NOM-199-SSA1-2000; las concentraciones de Pb en suelo de los hogares y talleres fueron comparadas con los límites máximos de la NOM-147-SEMARNAT/SSA1-2004 de 400 mg/kg (ppm);[36] y para las concentraciones de Pb en piezas se compararon con los límites máximos de la Comisión de Seguridad de Productos de Consumo de Estados Unidos (EE. UU.) de 100 ppm.[37] Para los valores que estuvieron por debajo del límite de detección (LOD) se consideró el valor del LOD de 5 ppm dividido entre dos. Se realizó una correlación de Spearman entre las concentraciones de Pb en sangre capilar, suelo y piezas, presentadas de manera gráfica, considerando un valor estadísticamente significativo $\leq$ 0.05. Además, se compararon las principales fuentes dietéticas de Ca y el consumo promedio diario con las recomendaciones de ingesta según las Guías Alimentarias para Mujeres Embarazadas Mexicanas.[38] Finalmente, se compararon las concentraciones de Pb en sangre capilar de acuerdo con el cumplimiento de la recomendación de consumo de Ca para mujeres embarazadas del Instituto de Medicina de EE. UU. (IOM)[39] aplicando la prueba U de Mann-Whitney, y se presentaron de manera gráfica. Todos los análisis se realizaron con el programa StataNow/BE 18.5 (StataCorp LLC, *College Station*, TX, USA).

RESULTADOS

En el ***(cuadro 1)*** se presentan las características generales de la población de estudio. La muestra estuvo compuesta por ocho mujeres embarazadas con una edad media de 24.9 años (± 4.4) y una EG promedio de 30 semanas (± 6.6), siendo en su mayoría primigestas (87%). El 62% de las participantes reportaron preparatoria como nivel máximo de escolaridad, todas las participantes fueron clasificadas con nivel socioeconómico bajo y el 38% presentó inseguridad alimentaria. En relación con su ocupación, el 62% reportó ser ama de casa, mientras que el 38% alfarera. El 25% reportó que su ocupación está relacionada con Pb, el 75% declaró tener un familiar que se dedica a la alfarería, así como tener un taller de producción doméstica de LBV dentro del hogar, respectivamente. En estos talleres el 25% de las participantes reportó el uso de PbO en la fabricación de las piezas producidas en su taller. Todas las participantes refirieron usar LBV para servir y/o almacenar alimentos.

Cuadro 1. Características generales de la población de estudio

Características	n=8
Edad (años)[a]	24.9 (4.4)
Edad gestacional (semanas)[a]	30.1 (6.6)
Embarazos previos[b]	
Primigestas	7 (87)
Nacimientos	1 (13)
Escolaridad[b]	
Primaria	1 (13)
Secundaria	2 (25)
Preparatoria	5 (62)
Nivel Socioeconómico[b]	
Bajo	8 (100)
Medio	0 (0)
Alto	0 (0)
Nivel de seguridad alimentaria[b]	
Inseguridad	3 (38)
Seguridad	5 (62)
Ocupación[b]	
Alfarera	3 (38)
Ama de casa	5 (62)
En su ocupación tiene contacto con Pb[b]	
Sí	2 (25)
No	6 (75)
Familiar alfarero[b]	
Sí	6 (75)
No	2 (25)
Uso de LBV para preparar o servir[b]	
Sí	8 (100)
No	0 (0)
Taller de alfarería dentro del hogar[b]	
Sí	6 (75)
No	2 (25)
Uso de esmalte con PbO en talleres[b]	
Sí	2 (25)
No	4 (50)
No aplica	2 (25)
Uso de suplementos actualmente[b]	
Sí	7 (87)
No	1 (13)
Tipo de suplementos[b]	
Calcio	1 (14)
Hierro	1 (14)
Multivitamínicos	2 (29)
Sulfato ferroso	3 (43)
Fuma o bebe alcohol actualmente[b]	
Sí	0 (0)
No	8 (100)
Fumadora pasiva[b]	
Sí	2 (25)
No	6 (75)
Presión sistólica (mmHg)[a]	102.8 (5.6)
Presión diastólica (mmHg)[a]	60.5 (8.1)
Peso (kg)[a]	66.3 (9.5)
Estatura (cm)[a]	156.1 (4.5)
IMC (kg/m^2)[a]	27.1 (3)

Pb: Plomo; IMC: Índice de Masa Corporal.a Los valores se presentan como media (DE). b Los valores se presentan como n (%)

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Cuadro 2. Concentraciones medias de Pb en sangre, suelo de los hogares y talleres y en piezas elaboradas en los talleres de las participantes y comparación con la normatividad vigente.

Pb en sangre capilar (µg/dL)	Pb en suelos de los hogares (ppm)	Pb en suelos de los talleres (ppm)	Pb en piezas (ppm)
31.4	72.3 (44.1)	160.8 (82.3)	149,707.8 (259,281.2)
16.3	N/A	N/A	N/A
14.2	195.6 (224.5)	121,523.9 (271,501.2)	139,061.5 (196,630.1)
13.2	N/A	N/A	N/A
12.4	8.7 (10.7)	43.3 (76.9)	<LOD
10.8	101.5 (126.4)	1,806.3 (2,089.1)	118.8 (111.2)
10.3	28.3 (26)	N/A	N/A
8.8	55.3 (46.3)	6 (6.1)	639.8 (901.2)
12.7 (9.6-15.6)a	34.5 (20-76)a	156.0 (13-318)a	23.0 (2.5-400)a

Pb: plomo. ID: número de identificación. LBV: Loza de barro vidriado. PbO: Óxido de plomo. CPS: Conteos por segundo. Ppm: partes por millón. LOD: Límite de detección. N/A: No aplica debido a que no se realizaron mediciones en los hogares y/o talleres. a Los valores se presentan como medina (p25, p75). Los valores resaltados en negritas superan el valor de referencia para mujeres embarazadas de Pb en sangre de 5 µg/dL establecido por la NOM-199-SSA1-2000; el límite máximo de Pb en suelos de 400 ppm establecido por la NOM-147-SEMARNAT/SSA1-2004 y; el límite máximo de 100 ppm de Pb en piezas de cerámica establecido por Comisión de Seguridad de Productos de Consumo de EE. UU. Las concentraciones de Pb en superficie se presentan con su media y desviación estándar.

Respecto al uso de suplementos, el 87% refirió usarlos, de los cuales los más consumidos fueron sulfato ferroso (43%) y multivitamínicos (29%). Ninguna mujer reportó el consumo actual de alcohol o tabaco, y el 25% informó convivir con un fumador (fumadoras pasivas). De acuerdo con las mediciones antropométricas, la presión arterial sistólica promedio fue de 102.8 mmHg (± 5.6) y 60.5 mmHg (± 8.1) para la presión diastólica, mientras que la media de IMC fue de 27.1 kg/m² (± 3).

En el ***(cuadro 2)*** se observa que la mediana de Pb en sangre capilar fue de 12.7 µg/dL (9.6-15.6), y todas las participantes superaron el valor de referencia de 5 µg/dL para mujeres embarazadas de Pb en sangre establecido por la NOM-199-SSA1-2000. Se observa, además, que se encontraron concentraciones de Pb en los suelos de los hogares; sin embargo, estas concentraciones no superan el límite máximo de 400 ppm establecido por la NOM-147-SEMARNAT/SSA1-2004. En el caso de las concentraciones de Pb en los suelos de los talleres, la mediana fue de 156.0 ppm (13-318). Se puede observar que dos de los cinco talleres evaluados superaron el límite máximo establecido por esta normatividad. Adicionalmente, encontramos que, en cuatro de los cinco talleres, las piezas producidas superan el límite máximo de 100 ppm establecido por la Comisión de Seguridad de Productos de Consumo de EE. UU.

En la ***(figura 1)*** se muestra la correlación entre las concentraciones de Pb de las mujeres embarazadas y las concentraciones de Pb obtenidas en los suelos de los hogares, suelo de los talleres y las piezas elaboradas en los talleres de las participantes. Se puede observar que existe una correlación estadísticamente significativa entre las concentraciones de Pb en sangre y las concentraciones de Pb en el suelo del hogar. Así como las concentraciones de Pb en suelo del hogar con las concentraciones de Pb en suelo de los talleres y en las piezas.

Sobre la ingesta de Ca de las participantes, en el ***(cuadro 3)***, se muestra que las principales fuentes de Ca son leche, yogurt, queso, tortillas, pescado, huevos y leguminosas, de las cuales únicamente el consumo de tortillas reportado por la población de estudio cumple con el número de porciones recomendadas por las Guías Alimentarias para Mujeres Embarazadas Mexicanas.

En la ***(figura 2)***, se puede observar que el 50% de las participantes (n = 4), cumple con la recomendación internacional de ingesta de Ca al día (≥ 1000 mg/d). Se observa que las mujeres que no cumplen las recomendaciones de Ca (< 1000 mg/d) tienen concentraciones más altas de Pb en sangre capilar con mediana de 12.8 µg/dL (11.5-14.8), en comparación con las que, si cumplen la recomendación con mediana de 12.5 µg/dL (9.8-22.8) de las mujeres que cumplen con la recomendación de Ca, sin embargo, no se encontró una diferencia estadísticamente significativa.

DISCUSIÓN

Este estudio descriptivo de mujeres embarazadas de una población alfarera del estado de Puebla muestra que la mediana de las concentraciones de Pb en sangre capilar es elevada 12.7 (9.6-15.6) µg/dL, y rebasa los valores de referencia para dicho grupo poblacional. Este resultado, es consistente con estudios previos que se han realizado en

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	Pb en sangre	Pb en suelo del hogar	Pb en suelo del taller	Pb en piezas
Pb en sangre	1.0			
Pb en suelo del hogar	**0.5831**	1.0		
Pb en suelo de taller	0.3138	**0.6015**	1.0	
Pb en piezas	-0.1585	**0.6277**	0.1769	1.0

Se realizó la prueba de correlación de Spearman. Los valores resaltados en negritas representan un valor de p = 0.001

Figura 1. Concentración de Pb en sangre capilar y su relación con fuentes relacionadas a la producción de LBV.

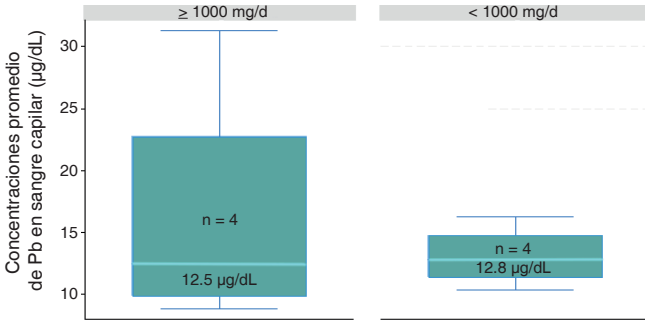

Figura 2. Concentraciones de Pb en sangre capilar de acuerdo con el cumplimiento de la recomendación de consumo de Ca para mujeres embarazadas.

poblaciones alfareras[40,41] mostrando niveles significativamente altos en comparación a la población general, a pesar de las iniciativas por producir barro libre de Pb. Sin embargo, nuestro estudio evalúa un grupo particularmente vulnerable, mujeres embarazadas que presentan exposición ocupacional y para-ocupacional a la "greta" u PbO.

En este estudio descriptivo, además, analizamos el Pb en el suelo de los hogares y los talleres, que frecuentemente se encuentran dentro de los patios de los hogares.[12] Observamos que, en general, las mujeres embarazadas viven o conviven en espacios altamente contaminados. El Pb en sangre capilar de la muestra analizada se correlacionó positiva y significativamente con el Pb en suelo de los hogares ($r = 0.58$, $p < 0.05$); a su vez, el Pb en el suelo correlacionó significativamente con el Pb medido en los talleres y en las piezas de LBV producidas. Anteriormente nuestro grupo de investigación realizó un análisis donde también observamos la correlación positiva y significativa entre el Pb en el taller y el nivel de sangre capilar en alfareros del estado de Morelos.[42] Cabe resaltar que los valores medidos en suelos y piezas de alfarería, en su mayoría, exceden los límites máximos permisibles por las normatividades, excediéndose hasta por cientos de veces.

Adicional a las mediciones de Pb, evaluamos características sociodemográficas, de las cuales, consideramos como relevantes la inseguridad alimentaria y el consumo adecuado de nutrimentos claves como el Ca, en este sentido un tercio de las mujeres viven con algún tipo de inseguridad alimentaria, prevalencia similar a estudios previos en zonas rurales de México.[43]

Una alimentación adecuada antes y durante la gestación constituye un determinante clave para la salud materna y neonatal.[44] El estado de nutrición materno influye no solo en el crecimiento y desarrollo fetal inmediato, sino también en procesos de programación fetal que pueden modificar el riesgo de enfermedades en etapas posteriores de la vida.[45]

La baja ingesta de Ca es particularmente preocupante en etapas de mayor demanda fisiológica, como en el embarazo.[46] Aunque las recomendaciones de ingesta de Ca durante el embarazo no se modifican de acuerdo con

Cuadro 3. Principales fuentes dietéticas de calcio (Ca), consumo promedio diario y comparación con las recomendaciones de consumo según las Guías Alimentarias para Mujeres Embarazadas Mexicanas

Alimentos fuente de Ca	Consumo promedio de Ca reportado por las participantes (g/día)	Tamaño de la porción recomendada (g o ml)	Número de porciones recomendadas al día	Número de porciones consumidas al día por las participantes
Leche*	184.3	240.0	2-3	0.8
Yogurt	44.2	180.0	2-3	0.2
Queso	30.8	40.0	2-3	0.8
Tortillas	181.9	30.0	6.5-8.5	**6.1**
Pescado	15.75	30.0	2-3	0.5
Huevos**	34.0	44.0	1-2	0.8
Leguminosas	67.0	120.0	2-3	0.6

Los valores resaltados en negritas son aquellas porciones consumidas al día que cumplen con la recomendación de consumo según las Guías Alimentarias para Mujeres Embarazadas Mexicanas.

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aquellas establecidas para mujeres en edad reproductiva,[39] este nutrimento adquiere especial relevancia en esta etapa debido a las adaptaciones fisiológicas que ocurren en el metabolismo ósea maternos para satisfacer las demandas del crecimiento y la mineralización fetal.[47] Por ello, la vigilancia de su consumo resulta fundamental. Una disponibilidad insuficiente de Ca no solo puede comprometer la salud ósea materna a mediano y largo plazo, sino también asociarse con desenlaces obstétricos adversos y con mecanismos biológicos que favorecen la absorción y movilización de otros elementos, incluidos metales pesados como el Pb.[48]

En este estudio observamos que más de la mitad de las mujeres embarazadas participantes no cubren las recomendaciones de Ca, en nuestra muestra se observó un consumo limitado de alimentos ricos en Ca, particularmente leche, yogurt, queso y pescados, lo que contribuye a la baja ingesta de este nutrimento. Estimaciones recientes indican que una proporción importante de la población global no alcanza las recomendaciones dietéticas de Ca, particularmente en países de ingresos bajos y medios.[49] En México el panorama no es distinto, el 54% de los adultos mexicanos presentan una ingesta inadecuada de Ca, similar a lo reportado en nuestra muestra.[50] En relación con los patrones dietéticos, se ha documentado que aquellos caracterizados por un alto consumo de leche descremada, productos lácteos, cereales integrales, pescado y otros mariscos, y con un bajo consumo de refrescos se correlacionó positivamente con un mayor consumo de Ca.[51]

En nuestro análisis, se muestra que hay mayores concentraciones de Pb en sangre capilar en aquellas mujeres que no cubren las recomendaciones de Ca en comparación con las que sí cumplen dicha recomendación (diferencia de 0.3 mg/dL, no significativa). Esto es consistente con estudios previos en embarazadas mexicanas donde se ha mostrado que la suplementación con Ca disminuye los niveles circulantes de Pb, ya sea por disminuir la absorción del metal pesado o por disminuir la liberación de Pb del hueso.[26]

Finalmente, los resultados de este estudio descriptivo no tienen poder estadístico suficiente ya que el tamaño de muestra es pequeño. Así como la presencia de un sesgo de selección al invitar únicamente a las mujeres que acuden al centro de salud comunitario, dejando fuera los casos graves de posibles efectos de intoxicación por Pb, pero, aunque el tamaño de la muestra es pequeño, nuestros resultados han reflejado de manera consistente las condiciones socioeconómicas reportadas en otros estudios de la población alfarera en nuestro país.

CONCLUSIÓN

Los resultados de este estudio evidencian una exposición elevada a Pb en mujeres embarazadas que residen en una comunidad alfarera, con concentraciones en sangre capilar que superan los valores de referencia establecidos para este grupo poblacional. La correlación observada entre el Pb en sangre y el Pb presente en los suelos domésticos y de los talleres, así como en las piezas de LBV, confirma que la producción artesanal de cerámica con esmaltes que contienen PbO constituye una fuente relevante de exposición tanto ocupacional como para-ocupacional. Estos hallazgos son especialmente preocupantes en el contexto del embarazo, debido al riesgo de efectos adversos en la salud materna y en el desarrollo fetal.

Adicionalmente, el estudio pone de manifiesto la coexistencia de exposición ambiental a Pb con condiciones de vulnerabilidad nutricional, particularmente una ingesta insuficiente de Ca. En conjunto, estos resultados subrayan la necesidad de estrategias integrales de salud pública que incluyan la eliminación progresiva del uso de Pb en la alfarería, el mejoramiento de las condiciones ambientales en los hogares y talleres, y el fortalecimiento de intervenciones nutricionales dirigidas a mujeres en edad reproductiva, especialmente durante el embarazo.

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CONFLICTO DE INTERÉS

Todos los autores declaran no tener conflicto de interés

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PROTOCOLOS

Abordaje interdisciplinario y protocolos de rescate en la extravasación de fármacos: una guía clínica.

Interdisciplinary Approach and Rescue Protocols in Drug Extravasation: A Clinical Guide

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RESUMEN

La extravasación de fármacos es una urgencia médica que ocurre cuando una infusión de sustancias irritantes o vesicantes se fuga hacia los tejidos subcutáneos. Este artículo presenta un protocolo estandarizado para su manejo, destacando que la colaboración entre médicos toxicólogos y enfermeros especializados en terapia de infusión es el factor determinante para minimizar lesiones de envergadura como la necrosis tisular o la pérdida funcional de la extremidad.

Palabras clave: extravasación, sustancias irritantes, infusión.

Abstract

Drug extravasation is a medical emergency that occurs when an infusion of irritant or vesicant substances leaks into the subcutaneous tissues. This article presents a standardized protocol for its management, highlighting that collaboration between medical toxicologists and specialized infusion therapy nurses is the determining factor in minimizing major injuries such as tissue necrosis or functional loss of the limb.

Key words: *extravasation, irritant substances, infusion.*

INTRODUCCIÓN Y SINTOMATOLOGÍA

La detección temprana es crucial. Los síntomas iniciales suelen ser dolor, ardor, hormigueo o quemazón en el sitio de punción. Al examen físico, puede observarse cambio de coloración (enrojecida, blanquecina o violácea), edema y tumefacción. En casos de agentes vesicantes, la formación de flictenas (ampollas), necrosis o ulceración puede manifestarse horas después del evento.

EL EQUIPO INTERDISCIPLINARIO: LA CLAVE DEL ÉXITO

El manejo efectivo de una extravasación requiere un equipo interdisciplinario entrenado. Los enfermeros especializados son la primera línea en la detección de signos de alarma y en la aplicación de técnicas de prevención, mientras que los médicos toxicólogos proporcionan el soporte farmacodinámico esencial para la indicación del antídoto correcto. Cualquier intervención con antídotos debe ser previamente consensuada con el servicio de toxicología para garantizar la seguridad del paciente.

CLASIFICACIÓN DE FÁRMACOS SEGÚN SU AGRESIVIDAD TISULAR

Los fármacos se clasifican según el daño que provocan al salir del torrente sanguíneo:

- Vesicantes: provocan necrosis tisular y pérdida del grosor de la piel (ej. citostáticos como antraciclinas o electrolitos como el gluconato de calcio).
- Irritantes (alto y bajo riesgo): causan dolor, quemazón y

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flebitis; los de alto riesgo pueden evolucionar a lesiones vesicantes.
- No irritantes: usualmente no causan daño, aunque grandes volúmenes pueden generar lesiones por efecto de masa.

PROTOCOLO DE ACTUACIÓN INMEDIATA

Ante una sospecha de extravasación, el equipo debe actuar bajo este orden de prioridades:
- Detener la infusión de inmediato.
- Aspirar a través del catéter entre 5-10 ml de sangre o líquido para extraer el fármaco remanente.
- Retirar el dispositivo después de la aspiración.
- Delinear la zona con marcador permanente, documentar con fotografías y registrar la hora.
- Inmovilizar y elevar la extremidad afectada sin generar presión.
- No aplicar vendajes compresivos ni punzar el miembro afectado para controles posteriores.

APLICACIÓN DE ANTÍDOTOS ESPECÍFICOS

El uso de antídotos debe seguir técnicas estériles y protocolos de dilución precisos:

1. Hialuronidasa (principalmente para electrolitos y no citostáticos)
   - Indicaciones: electrolitos (cloruro de potasio, bicarbonato de sodio, gluconato de calcio), nutrición parenteral total, aciclovir y dextrosa al 10-50%.
   - Aplicación: reconstituir 2000 UI en 10 ml de solución fisiológica. Se administran inyecciones subcutáneas de 0.2 ml en la periferia de la zona (tejido sano), espaciadas cada 2 cm.
   - Efecto: facilita la dispersión y absorción de la sustancia extravasada.

2. Dimetilsulfóxido (DMSO) 90-99% (específico para citostáticos)
   - Indicaciones: citostáticos como cisplatino, doxorrubicina, epirrubicina y mitomicina.
   - Aplicación: humedecer una gasa con 1-2 ml de DMSO y aplicarla sobre el doble del área afectada durante 15-20 minutos. Repetir cada 8 horas por 7 a 14 días.
   - Cuidados críticos:
     » Debe aplicarse solo sobre piel seca, ya que con agua genera una reacción exotérmica que causa quemaduras.
     » No aplicar sobre piel lesionada o volúmenes mayores a los indicados.
     » Informar al paciente que puede notar un olor a ajo en el aliento o alteraciones del gusto.
     » Si se requiere frío, aplicarlo siempre después del DMSO.

3. Fentolamina
   - Indicaciones: inotrópicos (adrenalina, noradrenalina, dopamina) y ciertos citostáticos (vincristina, vinblastina, paclitaxel).
   - Aplicación: diluir 5 mg en 10 ml de solución fisiológica. Administrar mediante inyecciones subcutáneas en la circunferencia de la lesión (0.1-0.2 mg/kg, máx 5 mg).

CUIDADOS POSTERIORES Y CONCLUSIÓN

Tras el rescate, se debe evitar el baño de inmersión por 24 horas, no cubrir la zona con ungüentos no indicados y mantener la vigilancia clínica de la evolución cutánea. La formación continua del personal y la existencia de guías institucionales son la mejor herramienta para transformar un evento adverso potencial en un incidente controlado con mínimas consecuencias para el paciente. Es crucial no colocar accesos vasculares ni utilizar el miembro afectado para toma de presión. La clave esta en el trabajo interdisciplinario de evaluación del plan farmacológico y el acceso adecuado para ese fin. Una buena comunicación del equipo dará lugar a la selección correcta del dispositivo de infusión, una detección adecuada permitirá tratar las lesiones antes de que se generen ulceras o necrosis, el seguimiento posterior es clave.

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2nd International Congress EnviroEpiHealth[MX]

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PREFACE

Exposures that, directly or indirectly, cause chronic diseases often occur years, even decades, before they are diagnosed. Therefore, biomarkers collected at specific points in time do not help reveal the full story of how a disease develops in an individual. For that, it is necessary to consider the collection of exposures experienced throughout an individual's lifetime (exposome). The term "exposome" was coined in 2005 by Christopher Wild and reflects the reality of people's exposure to potentially harmful agents from both polluting and non-polluting sources, including industrial chemicals, combustion emissions, radiation, heat/cold, noise, and food. The exposome also includes behavioral factors, such as activity levels and stress responses. Finally, an individual's exposome includes their microbiome, or a vast, personalized community of commensal microbes. All of these exposures and factors can vary throughout a single day, let alone the weeks, months, and years that make up a lifetime. In conclusion, a person's exposome is the sum total of exposure to many factors that fill the days, months, and decades of that person's life: exposure to chemicals, radiation, heat/cold, noise, food, stress, and other environmental agents; health behaviors and activities; and the unique profile of commensal bacteria that make an individual who they are.

At the Second International EnviroEpiHealth Congress, held in Puebla at the Julio Glockner Auditorium of the BUAP Faculty of Medicine from October 6-9, 2025, oral presentations and posters on scientific dissemination and research were presented on topics such as the effects of air pollution, atmospheric pollution, obesity, environmental toxicology, epigenetic alterations (such as DNA methylation, modifications in chromatin structure, transcriptional modifications, and liquid-liquid phase separation molecular biocondensates), microbiota and microbiome, and altered metabolic pathways associated with chronic diseases, which could favor a pathological phenotype in various contexts.

The abstracts are reproduced as accepted by the organizing committee. The scientific sessions are listed as follows:

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CC 1. *Health and Environment*

María Lilia Cedillo Ramírez
Benemérita Universidad Autónoma de Puebla, México. Rectora

Epigenetics regulates gene expression without altering the DNA sequence, through processes such as DNA methylation, histone modifications, and the action of non-coding RNAs. These mechanisms respond to environmental signals, such as nutritional, microbial, and inflammatory cues, making them a point of integration between genetics and lifestyle. In this context, microbial metabolites, particularly short-chain fatty acids, can directly modulate chromatin structure and influence inflammatory and metabolic processes. The APOE gene plays a role, with its APOE3 and APOE4 isoforms exhibiting differential effects on lipid homeostasis. APOE3 is associated with a more stable metabolism, while APOE4 increases vulnerability to dyslipidemia, elevating LDL and promoting oxidative stress. The role of environmental signals is evident in the Mediterranean diet, whose high content of fiber, monounsaturated fats, and antioxidants helps modulate the microbiota, improve the lipid profile and promote healthier epigenetic markers, partially mitigating the adverse effects associated with APOE4.

CC 2. *Gene–Environment Interactions: Challenges and Opportunities for Precision Medicine in the Post-Genomic Era*

Erick I. Navarro Delgado
University of British Columbia, Canadá.

The post-genomic era has revealed a complex landscape in which genetic variation alone cannot fully explain a substantial number of health and disease phenotypes. There is growing evidence of the critical role of gene-environment (GxE) interactions in shaping individual risk profiles, therapeutic responses, and disease trajectories. Precision medicine will benefit significantly from integrating GxE insights, enabling more personalized risk assessments, earlier interventions, and individualized treatment strategies. This talk explored the conceptual and methodological challenges of studying GxE in the field of omics, including complex modeling requirements, measuring environmental exposure, population diversity, and replicability. Despite these difficulties, advances in multi-omics technologies, large cohort studies, interdisciplinary collaborations, and machine learning methods offer new avenues for unraveling the intricate interplay between genetic predisposition and environmental influences. By harnessing the power of omics data in genetically and socioeconomically diverse studies, the GxE paradigm offers a promising field for understanding complex biological traits and developing transformative healthcare strategies.

CC 3. *Influence of Adipocytes and the Microenvironment on Epigenetic and Translational Reprogramming in Cancer*

Karla Tovar-Hernández,[1,2] Yarely M. Salinas-Vera,[1] César López Camarillo.
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Recent epidemiological evidence has linked obesity to an increased risk of developing breast cancer. Breast adipocytes interact with cancer cells, inducing the progression of breast tumors; however, the molecular mechanisms remain poorly understood. In vitro three-dimensional (3D) cultures of cancer cells better mimic changes in gene expression in in vivo tumors and represent an innovative experimental approach in the study of cancer biology. In the present study, we analyzed the effects of adipocytes on the proteome of Hs578t breast cancer cells grown in a 3D microenvironment. The results of proteomic analyses using LC-MS/MS mass spectrometry demonstrate that 87 proteins increased their expression in Hs578t cells grown in the presence of adipocytes for 48 h, while 224 were repressed under the same conditions ($p<0.05$, FC>2.0). Bioinformatics analyses showed that some of the overexpressed proteins, such as PITPNM2, ATP2C1, ABCA12, HDLBP, and APOB, have functions related to lipid transport and localization. High expression of these factors was associated with low survival in breast cancer patients. Other proteins identified as being regulated participate in tumor hypoxia, differentiation, and cancer progression. Through functional studies, we found that blocking apolipoprotein B (APOB) expression by siRNAs in Hs578t cells grown with adipocytes resulted in a decrease in the size of 3D cell structures and a decrease in intracellular lipid concentration. In conclusion, the data suggest that the 3D microenvironment and communication with adipocytes reprogram the proteome of breast cancer cells. These data help us understand the effects of the environment on gene expression and contribute to the discovery of new tumor proteins with potential applications in breast cancer therapy.

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CC 4. *Don't Entertain the Baby or Grandparent With Your Cell Phone!*

Arturo Ortega Soto
CINVESTAV, México

In their recent study, Dr. Arturo Ortega Soto and colleagues analyzed how Müller cells (the main glial cells of the retina) participate in glutamate clearance through specialized transporters. These transporters, essential for preventing excitotoxicity, exhibit activity that changes throughout the day-night cycle, suggesting circadian regulation of the glutamate recycling system. The use of cell phones and tablets to entertain infants or the elderly could disrupt delicate neurotransmission dynamics and circadian regulation in the retina, especially if it occurs at inappropriate times or with high intensities of blue light. To better understand these effects, it is essential to continue with experimental and clinical studies that examine glutamate transport mechanisms, the glial response, and light-induced changes in vulnerable populations.

CC 5. *Chronic Radon Exposure and Its Impact on Thyroid Cancer*

Carlos Arturo González Castañeda
FACMED- BUAP, México

Galicia is located in the northwest of the Iberian Peninsula, bordered to the north by the Cantabrian Sea and to the west by the Atlantic Ocean. In Galicia, many houses and buildings are constructed with Galician granite, a rock abundant in the region. This material, although resistant and iconic, contains natural uranium that releases radon gas, making Galicia one of the areas with the highest exposure to this gas in Europe, with health implications (such as an increased risk of lung cancer and, to a lesser extent, possible effects on the thyroid). Although the link between the development of lung cancer and radon inhalation is well known, cases of thyroid cancer appear to be increasing in Galicia. Work carried out by INIBIC and CHUAC in Galicia on a cohort of patients seen between 2014 and 2015 linked these cases to radon exposure, given the circumstances of their housing. To analyze the impact of this on some patients, a comet assay was performed. The comet assay detects single- and double-strand breaks in DNA and other types of genomic damage. The name "comet" comes from the appearance of cell nuclei after electrophoresis: a head (intact DNA) and a tail (damaged fragments that migrate). In its initial phases, the project demonstrated a possible relationship that there was a possible relationship between radon and the formation of thyroid neoplasms; this relationship would have to be subjected to more rigorous tests, for which purpose the project was initiated and later presented to the Xunta de Galicia to begin a broader and deeper analysis on the subject.

CC 6. *Preconception Paternal Health for Healthy Offspring*

Juan Carlos Flores Alonso
CIBIOR-IMSS, México

The aim of this presentation was to break the stigma that a baby's health depends 100% on the mother's womb. Dr. Flores Alonso emphasized that sperm is much more than a simple DNA carrier; it is a complex messenger that carries information about the father's environment. Considering several important points, we have: 1) The Impact of Epigenetics: the key concept here is fetal programming of paternal origin, where a man's lifestyle affects the chemical marks on his sperm's DNA. The consequences of poor habits in the father can include sending "wrong" signals to the embryo, programming its metabolism incorrectly even before the mother knows she is pregnant. 2) Lifestyle Factors and Their Effects: the testicular microenvironment is sensitive to oxidative stress caused by poor diet and a sedentary lifestyle, which damages sperm membranes. 3) Toxic Exposures: Alcohol and tobacco consumption not only decrease sperm count but also alter the levels of messenger RNA that sperm delivers to the egg. 4) Metabolic Syndrome: A father with insulin resistance or who is overweight can pass on to his offspring a genetic predisposition to the same conditions (transgenerational inheritance). Window of Opportunity: One of the most practical points is timing. Since the spermatogenesis cycle (the creation of new sperm) lasts approximately 72 to 74 days, the father has a "window of opportunity" of about 3 months to improve his health and, therefore, the quality of the genetic information he will transmit. Main Recommendations: The importance of antioxidants (such as folic acid, zinc, and vitamin E) in men is often mentioned. Shared Responsibility: Preconception health is a couple's responsibility. Medical Evaluation: Men should undergo checkups to detect infections or hormonal imbalances before conception. In conclusion, preventing chronic diseases in the child's adult life begins with the father's health months before conception. "Healthy parents, healthy children" is not just wishful thinking, but a documented biological reality.

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CC 7. *From Patient to Laboratory: Foundations for the Collection of Clinical Data and Biological Samples in Translational Science*

Erika Chavira Suárez
Facultad de Medicina-UNAM, México

Translational science seeks to integrate basic knowledge with clinical practice to generate innovative healthcare solutions. In this context, the proper collection, handling, and storage of both clinical data and biological samples is essential to ensuring the validity and reproducibility of research. This talk was designed to provide students, researchers, and healthcare professionals with a comprehensive overview of the processes that connect the patient to the laboratory. It reviewed the methodological and ethical principles related to obtaining clinical information and biological samples, as well as the quality standards necessary to ensure their traceability and usability in subsequent studies, including those related to the exposome. Through theoretical and practical sessions, topics such as informed consent and ethical data protection, procedures for collecting blood, tissues, and other biological fluids, clinical data standardization strategies, and logistical considerations for biobanks will be addressed. Practical case studies will also be discussed, illustrating how proper sample and data management impacts the generation of translational knowledge aimed at improving disease prevention, diagnosis, and treatment. This talk sought to strengthen participants' skills in bridging the critical gap between clinical practice and laboratory research, fostering the development of multidisciplinary teams capable of addressing the challenges of translational research and exposome studies in Mexico.

CC 8. *From Air to Cell With Rothman's Causal Model: An Epidemiological Perspective on Diseases Associated With Air Pollution*

Sagrario Lobato Huerta
Secretaría de Salud del Estado de Puebla, México

Air pollution, particularly exposure to fine particulate matter (PM2.5), has been consistently linked to various adverse health effects, including cardiometabolic disorders, respiratory diseases, obesity, and perinatal complications. However, a knowledge gap persists regarding the weight and hierarchy of the etiological factors that contribute to these conditions. The Bradford-Hill causality model, widely used in epidemiology, allows for the identification of associations but does not clearly distinguish the type or strength of the causes involved, thus limiting the generation of robust evidence to guide public health interventions. The Rothman causal model offers an alternative by classifying sufficient causes and components, which fosters a more comprehensive view of the multifactorial nature of diseases associated with air pollution. This talk explored its application as a conceptual and methodological tool to strengthen causal inference and support more effective environmental health strategies.

CC 9. *Epigenetic Mechanisms in the Development and Progression of Liver Cancer*

Denisse Cruz Trejo
USEP. Puebla, México

Liver cancer and cirrhosis represent a serious public health problem due to their high morbidity and mortality. Early detection is difficult, limiting intervention opportunities and contributing to unfavorable clinical outcomes. In this context, analysis of the epigenetic profile of hepatocellular carcinoma has identified key modifications, including global hypomethylation of oncogenes and hypermethylation of tumor suppressor genes, histone changes that alter chromatin accessibility, and regulation via microRNAs (miRNAs). This talk offers a comprehensive view of these epigenetic marks, highlighting their potential as biomarkers and possible therapeutic targets for the development of more personalized and effective treatment strategies.

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CC 10. *Drug Design in the 21st Century: Towards Computer-Assisted Automation*

Edgar López
CINVESTAV, México.

Computer-aided drug design (CADD) has become a cornerstone of modern biomedical research, accelerating the discovery and optimization of bioactive molecules. This talk introduced fundamental in silico strategies, including ligand-based, structure-based, and consensus approaches, highlighting their complementarity within rational drug design. Special emphasis was placed on CADD applications in epigenetic drug discovery, a rapidly growing field with significant therapeutic implications for chronic diseases. Through molecular modeling, virtual screening, and protein-ligand interaction prediction, it is now possible to identify modulators of key epigenetic enzymes—such as DNMTs, HDACs, and bromodomains—while simultaneously improving selectivity and safety profiles. Emerging trends were also discussed, including expanding the chemical space through virtual libraries, artificial intelligence for bioactivity prediction, in silico polypharmacology for multitarget drug design, and workflow automation. Finally, the cross-cutting impact of CADD was framed within the One Health paradigm, highlighting its role in human, animal and environmental health.

CC 11. *Thinking About Future Generations: The Importance of Taking Care of Our Eggs*

Isabel Hernández Ochoa
CINVESTAV. México

Female fertility has declined in recent decades, and mounting evidence suggests that exposure to toxic compounds plays a significant role in this trend. In modern life, we are constantly surrounded by a wide range of products—plastics, cosmetics, pesticides, cleaning agents, food additives, and industrial materials—that release chemicals capable of disrupting normal biological processes. Many of these compounds are classified as endocrine disruptors, meaning they can interfere with hormonal signaling pathways that are essential for reproductive health. Despite their widespread presence, the long-term consequences of these exposures remain poorly understood, particularly regarding their impact on future generations. This talk explored the adverse effects associated with exposure to commonly used toxic compounds on ovarian function, with a particular focus on oocyte development and quality. The oocyte is highly sensitive to environmental stressors; Even exposure to low doses during critical developmental windows can alter mitochondrial function, induce oxidative stress, or disrupt meiosis, ultimately affecting fertility outcomes. Recent research also suggests that such exposures can not only affect a woman's reproductive capacity but may also have transgenerational effects, potentially influencing embryonic development, offspring health, and even the reproductive potential of subsequent generations. Understanding how everyday chemicals affect ovarian biology is essential for improving reproductive health strategies, informing public policy, and promoting safer environments. By identifying the mechanisms by which these toxic compounds act, we can better assess the risks they pose and work to reduce their impact on fertility and long-term health.

CC 12. *Telomere Length and Environment During the First Years of Life*

Citlalli Osorio Yáñez
Instituto de Investigaciones Biomédicas, UNAM, México

Telomeres are guanine (G)-rich DNA sequences responsible for protecting chromosomes and signaling the end of replication in eukaryotes. Each time a cell divides, telomeres shorten, and therefore telomere shortening has been used as a biomarker of cellular aging. Furthermore, due to their high G content, telomeres are susceptible to damage from oxidative stress. Telomere shortening is not a constant process throughout life, as accelerated telomere shortening has been demonstrated in the first four years of life. Other factors, such as the telomere length of the mother and father, determine the telomere length of the newborn. Additionally, factors such as violence and unfavorable socioeconomic and stressful conditions have been associated with telomere shortening in childhood. Regarding environmental pollutants, studies have shown that exposure to tobacco smoke, metals, air pollution, and plasticizers can shorten telomere length in early life. Reconsidering our consumption habits and reducing, as much as possible, our exposure to environmental toxins, especially among young children, is essential for protecting future generations.

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CC 13. *Social Processes and Epigenesis: Dimensions Associated With Quality of Life in the Context of Socio-Environmental Devastation of the Upper Atoyac*

Alfredo Delgado Rodríguez
Centro de Investigaciones Interdisciplinarias sobre Desarrollo Regional, UATx, México

This talk presented a comprehensive reflection on social processes and epigenesis as fundamental dimensions for understanding quality of life in the context of the socio-environmental devastation of the Upper Atoyac, a region encompassing territories in Puebla and Tlaxcala. Building on Dr. Delgado's previous work on well-being, socio-environmental justice, and violated ecologies, Dr. Delgado argued that the degradation of the Atoyac River cannot be analyzed solely through physicochemical parameters; on the contrary, it constitutes a complex phenomenon in which histories of territorial dispossession, accelerated industrialization, persistent inequality, and everyday practices converge, revealing how bodies and territories co-constitute each other. Dr. Delgado proposed understanding social epigenesis as a process through which environmental devastation leaves not only ecological traces, but also cultural, affective, and biopolitical ones. Riverside communities experience continuous exposure to environmental risks, but also to processes of institutional silencing, deterioration of the social fabric, and a redefinition of their life horizons. This historical accumulation of violence is inscribed on bodies, shaping health, risk perceptions, ways of inhabiting the land, and the possibilities for agency. Dr. Delgado presented elements derived from collaborative research in the region, which identifies practices of community resistance, territorial reappropriation, and the construction of possible futures. Dr. Delgado argued that the transformation of the Upper Atoyac requires recognizing the interdependence between social, epigenetic, and political processes, as well as promoting community-based agendas for socio-environmental justice that allow for the reconstruction not only of the ecosystem, but also the conditions for a dignified life.

CC 14. *Public and Environmental Health: Health Effects and Multisectoral Management in Mexico*

Elena Parra Tapia
Secretaría de Salud, México

Air pollution is the leading environmental risk to public health worldwide and a major cause of morbidity and mortality in Mexico, ranking among the top risk factors for death. In 2021, more than 50,000 deaths were attributed to air pollution, largely due to chronic and acute exposure to pollutants such as PM2.5, ozone, and nitrogen dioxide. PM2.5 is particularly harmful because it can penetrate deep into the lungs and enter systemic circulation. Studies in Mexico have shown that increases of 10 µg/m³ in PM2.5 are associated with higher mortality from cardiovascular, respiratory, and other systemic diseases, affecting all population groups. Air pollution also disproportionately impacts vulnerable populations, including children and pregnant women, contributing to adverse outcomes such as premature birth and neurodevelopmental disorders. Several air pollutants, including PM2.5, are classified as carcinogenic to humans.
Major sources of air pollution in Mexico include vehicular traffic, industry, power generation, domestic fuel use, agriculture, and forest fires. While regulatory actions have improved air quality in large cities, progress has been uneven, with persistent regional inequalities. To address this, Mexico has implemented a multisectoral management approach and developed an Epidemiological Surveillance System for Health Damage from Environmental Pollution, which integrates environmental and health data to support risk identification and decision-making. Although air pollution remains a critical public health challenge with high social and economic costs, strengthened regulation, surveillance, and intersectoral collaboration provide a strong foundation for reducing health impacts and protecting vulnerable populations.

CC 16. *Exposome and HPV: When Interactions Also Write Evolution*

Noé Velázquez Márquez
Departamento de Biología Celular, BUAP, México

Human papillomavirus (HPV) does not evolve in isolation: it develops within the web of exposures that constitutes the exposome throughout life. Emerging evidence demonstrates that the cervicovaginal microbiome influences viral fate. Chemical and behavioral exposures, particularly tobacco use, and endocrine-nutritional contexts can erode antibody quality and mucosal immunity, while chronic immunological states such as HIV alter the magnitude and durability of vaccine responses. At the same time, antigenic diversity at the L1/L2 lineage level implies that HPV16 is not immunologically monolithic; therefore, the immune landscapes shaped by vaccination and local exposomes generate spatially heterogeneous selection pressures. Population data confirm a substantial vaccine impact without

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a consistent signal of genotype replacement; however, they underscore the need for continuous genomic and phylodynamic surveillance tailored to type and lineage. This perspective advocates for the integration of exposomal metrics into risk stratification and program evaluation, as well as the development of broader antigen designs to ensure long-term protection in variable environments.

CC 17. *Genetics and Radiotherapy in Cancer*

Ma. Dolores de la Mata Moya
Centro Médico ABC, México

In her talk, Dr. María Dolores De la Mata discussed how genomic variations influence tumor radiosensitivity and the risk of adverse effects in healthy tissues during radiotherapy. She explained that genetic differences affecting DNA repair, cellular stress responses, and inflammation help determine why tumors respond differently to radiation and why some patients experience greater treatment-related toxicity. She highlighted the growing role of personalized medicine in radiation oncology, emphasizing that incorporating genetic information into treatment planning can optimize therapeutic effectiveness while minimizing side effects, thereby improving patients' quality of life. Dr. De la Mata also integrated perspectives from molecular genetics, epidemiology, and environmental factors, stressing that cancer development and treatment response result from the interaction between genetic susceptibility and environmental exposures. This integrated approach is essential for both cancer prevention and more effective therapies. Finally, she emphasized the need for interdisciplinary research, bringing together clinicians, geneticists, and public health scientists to advance toward safer, more precise, and more effective cancer treatments.

CC 18. *The 2030 Agenda at the University and How It Influences SDG 3: Good Health and Well-Being*

Josué Gómez Nava
ManUp Campaign, ECOSOC, Naciones Unidas Nueva York, Red Internacional de Promotores ODS, México/USA

This talk addressed the integration of the UN's 2030 Agenda, specifically Sustainable Development Goal (SDG) 3: Good Health and Well-being, into the work of higher education institutions. The central argument was that universities must transcend their traditional role to become strategic actors and drivers of social change, aligning their core functions to promote sustainability. The talk defined SDG 3 not only as a hospital health goal, but as a universal humanitarian promise that demands comprehensive care, encompassing healthy environments, disease prevention, and coverage for all ages. Universities play a crucial role through three key areas: training professionals capable of implementing health policies, conducting applied research to solve local and global health problems, and promoting university outreach through community-based programs. The presentation underscored the mutual benefit of this commitment: the SDGs provide universities with a global framework for defining the "responsible university," while academia offers solutions, knowledge, and innovative ideas, training the future leaders and implementers of the 2030 Agenda. However, the document acknowledges significant challenges, such as limited funding and the need for greater inter-institutional coordination. This is contextualized with local data, such as the high rate of poverty in Puebla, which underscores the urgency of ensuring equitable access to healthcare services. In conclusion, the main message was that the university of the future will be one that integrates health and well-being as cross-cutting themes in all its activities, functioning as a true ecosystem for social transformation. The university's ethical commitment to public health is key to building a more just, equitable, and sustainable society.

CC 19. *Food From Space to the World: Food Security*

Enrique Antonio Álvarez Díaz,
CEO Vidaliz, México

The Mexican company Vidalis, renowned for developing foods with an extended shelf life, is applying its technology to global food security, inspired by the requirements of space missions. The company's founder and leader, Enrique Álvarez Díaz, presented the paper "Food from Space to the World: Food Security," highlighting his recent, already patented invention: a food preservation technique. His presentation focused on Vidalis, a company dedicated to food innovation that offers nutritious, long-lasting, and practical solutions for both everyday consumption and demanding needs such as space exploration and emergency preparedness.

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CC. 20 *Fetal Programming and Epigenetic Modifications*

Alonso Antonio Collantes Gutiérrez
Hospital Universitario BUAP, México

Fetal programming allows for adaptations to the intrauterine environment that, in the long term, can increase the risk of chronic diseases. These effects originate primarily from epigenetic mechanisms, such as DNA methylation, established during critical stages of development. Dr. Collantes presented information on how maternal malnutrition or obesity during pregnancy alters fetal metabolism and predisposes individuals to metabolic diseases. He also highlighted that epigenetic changes in sperm, particularly the genetic imprinting of IGF2 and PEG3, can transmit metabolic risk to offspring. He mentioned the EARNEST project, focused on Omega-3 supplementation in early childhood, as a strategy to modulate epigenetic processes and improve future health. Finally, he addressed the relationship between fetal programming and cancer, highlighting the hypermethylation of tumor suppressor genes such as RASSF1A and alterations in key genes (TP53, BRCA1, MYC, KRAS) in low birth weight or preterm fetuses. He concluded by emphasizing the need for public health actions, such as nutritional education for women of reproductive age and metabolic assessment protocols during pregnancy.

CC 21. *The Consumption of Psychoactive Substances and Their Epigenetic Effects on Male Germ Cells*

Yessica Ibelith Cosme Herrera
Universidad de la Salud del Estado de Puebla, México

The use of psychoactive substances during the male reproductive years constitutes an epigenetic factor with direct implications for sperm quality and the molecular programming of offspring. Substances such as ethanol, nicotine, Δ⁹-tetrahydrocannabinol (THC), and benzoylecgonine cross the blood-testis barrier, altering key spermatogenesis processes through epigenetic mechanisms that include DNA methylation, post-translational histone modifications, and microRNA dysregulation. These alterations are mediated by regulatory enzymes such as DNA methyltransferases (DNMT1, DNMT3A, DNMT3B), TET dioxygenases (TET1-3), histone deacetylases (HDACs), acetyltransferases (HATs), lysine demethylases (KDMs), and sirtuins (SIRT1 and SIRT6). Ethanol promotes oxidative stress and interferes with DNMTs and TETs, leading to hypomethylation of BDNF and GNAS and hypermethylation of MEST and LINE-1. Nicotine negatively modulates HDACs and DNMTs, altering genes such as PGAM5, PTPRN2, TYRO3, and P16. Cocaine decreases the activity of TETs and KDMs, affects Cdkn1a, and promotes histone acetylation. Cannabis interferes with cAMP/PKA signaling through CB1/CB2 cannabinoid receptors, affecting Dlgap2 methylation. Consequently, sperm DNA fragmentation, decreased motility, altered protamine/histone ratios, and abnormal expression of microRNAs such as miR-21, miR-30, and miR-142 are observed. These epigenetic marks can be inherited by offspring, even in the absence of direct exposure, generating metabolic, neurological, immunological, and behavioral effects. These findings reinforce the need to include the paternal dimension in reproductive health and in the study of epigenetic mechanisms with transgenerational effects.

CC 22. *Three-Dimensional Genomic Aberrations in Lung Diseases*

Guillermo Barreto
Université de Lorraine, IMoPa Institute, EPIGLYCAN Laboratory, Francia

Today, the importance of the genome's 3D architecture (TADs, A/B compartments, and enhancer-promoter interactions) as an essential regulator of gene expression is undeniable. This talk highlighted that in lung diseases, especially cancer and fibrosis, these structures can be reorganized by mutations, copy number variations, or chromosomal rearrangements, altering the physical proximity between genes and their regulatory elements. Hi-C analysis reveals a restructuring of compartments and connectivity between distal regions, associated with transcriptional changes and genomic variations. In pulmonary fibrosis, architectural proteins such as HMGA2 can induce chromatin openings and require H2A.X phosphorylation to enable demethylation processes that activate profibrotic pathways. Several risk variants identified by GWAS for diseases such as COPD are located in enhancers and promoters whose activity depends on the spatial context of the genome. Integrating chromatin accessibility and 3D mapping has enabled the prioritization of mutations that disrupt critical regulatory interactions. Three-dimensional aberrations not only trigger a pathophysiological process but can also accelerate it, making spatial epigenetics an emerging therapeutic target in lung diseases.

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CC 23. *The Exposome and the Developmental Origins of Health and Disease (DOHaD): The Relationship Between the Environment and Health in Early Life*

Ericka Chavira Suárez
Facultad de Medicina, UNAM, México

Within the Developmental Origins of Health and Disease (DOHaD) paradigm, early childhood exposures, including nutrition, pollutants, psychosocial stress, and the microbiome, play a critical role in shaping long-term health trajectories and disease susceptibility. Integrating exposome science with DOHaD research provides unique opportunities to identify critical periods of vulnerability, develop biomarkers of exposure and effect, and inform preventive strategies tailored to maternal, fetal, and infant health. A key example is placental complications of pregnancy, such as preeclampsia, intrauterine growth restriction, and gestational diabetes, which are strongly influenced by maternal environmental exposures and contribute significantly to perinatal morbidity and mortality worldwide. Globally, these complications pose a significant challenge to maternal-fetal medicine, with profound implications for both immediate neonatal outcomes and lifelong health risks. In Mexico, their prevalence is particularly concerning, reflecting the combined impact of socioeconomic disparities, nutritional transitions, high rates of obesity and metabolic disorders, and limited access to specialized prenatal care. Integrating global and Mexican perspectives underscores the urgent need to apply exposome-based Origins of Health and Disease (DOHaD) research to better understand how the environment influences placental function and early developmental programming. This approach not only drives risk assessment and biomarker discovery but also guides context-specific preventive interventions aimed at reducing maternal and child health inequities. By promoting multidisciplinary strategies that integrate epidemiology, molecular biology, and systems science, DOHaD exposome-based research has the potential to transform public health policies and foster healthier life trajectories in all populations.

CC 24. *Genomics and the Exposome: An Integrative Analysis to Understand Epigenomic Differences at the Population Level*

Erick I. Navarro Delgado
University of British Columbia, Canadá

DNA methylation (mDNA) is an epigenetic mark involved in chromatin structure and gene regulation, associated with various health outcomes. While interindividual mDNA variability has been primarily linked to genetic variants (G) and environmental exposures (E), it is still uncertain where in the genome these factors might influence mDNA, or whether they act individually, additively (G+E), or interactively (GxE). To address this knowledge gap, The speaker presented RAMEN (github.com/ErickNavarroD/RAMEN), an R package that provides the first localizable, accessible, interoperable, and reusable framework for estimating the contributions of the genome and exposome to methylome variability in microarrays. RAMEN uses machine learning and statistical techniques to identify the best explanatory model (G, E, G+E, GxE, or None) of variable methylated loci (VMLs; genomic regions with high mDNA variability), controlling for spurious associations. To understand the contribution of gene-environment interaction to the DNA methylome during fetal development, we applied RAMEN to paired umbilical cord blood and placental samples from 657 newborns in the CANDLE study, a socioeconomically diverse cohort with extensive prenatal genomic, methylomic, and exposome characterization (approximately 3 million SNPs, approximately 800,000 CpGs, and 203 exposures). A more variable and distinctive DNA methylome in the placenta compared to umbilical cord blood has been observed. Furthermore, genetics emerged as a key factor in DNA methylome variability, frequently in combination with prenatal exposures (Umbilical cord blood: G=10.2%, G+E=12.4%, GxE=11.5%, E=0.1%, N=65.8%; Placenta: G=15.8%, G+E=12.3%, GxE=7.2%, E<0.1%, N=64.7%). Genetic factors explained a greater amount of partitioned variance compared to environmental and interaction factors. These findings contribute to understanding how and where, in the genome, genetics and environmental factors can interact to influence DNA methylation.

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CC 25. *Relevance of Environmental Risk Studies for Health*

María Del Rocío López Vargas
Escuela Nacional de Ciencias de la Tierra, México

The environmental health risk situation in Mexico is characterized by increasing complexity stemming from rapid industrialization, deficiencies in the regulation and monitoring of chemical emissions and discharges, and persistent social inequalities that amplify exposure to pollutants. Several regions of the country, including the central industrial corridors, the Upper Atoyac and Lerma-Santiago river basins, and the northern mining districts, exhibit significant levels of risk due to the presence of toxic metals, volatile organic compounds, particulate matter, industrial discharges, and agrochemicals. Environmental risk analysis allows for the identification of vulnerable populations, exposure pathways, and potential harms that are not always visible through conventional epidemiological surveillance. Available evidence shows associations between environmental pollution and increases in respiratory, cardiovascular, neurological, and reproductive diseases, as well as certain types of cancer, disproportionately affecting communities with fewer resources and less access to healthcare services. Despite progress in environmental regulations and monitoring, Mexico faces challenges such as institutional fragmentation, limited inspection capacity, a lack of systematic human biomonitoring, and poor coordination among environmental, health, and community authorities.
Strengthening the application and monitoring of environmental risk assessments in the country is essential for generating accurate diagnoses, guiding preventive public policies, and promoting interventions based on socio-environmental justice. In a country where environmental degradation directly impacts quality of life, addressing these risks is crucial to guaranteeing the constitutional right to a healthy environment.

CC 26. *Environmental Exposure to Microplastics: Occurrence and Health Implications in the Eastern Region of México*

Arely Anaya Hernández
Laboratorio de Toxicología y Química Ambiental Centro de Investigación en Genética y Ambiente Universidad Autónoma de Tlaxcala

Microplastic pollution (MPs; plastic fragments <5 mm) represents an emerging problem with environmental and human health repercussions. In eastern Mexico, particularly in Tlaxcala and Puebla, its presence in water, bioindicator fauna, and human fluids was evaluated using a One Health approach. In aquatic environments, all surface water bodies analyzed, including springs and lagoons, contained MPs, predominantly films and fibers, with higher concentrations in urban water bodies, demonstrating a constant source of exposure for both the biota and the population consuming water from these sources. Additionally, bottled water from both states was evaluated, finding MPs in all samples, suggesting that even treated and commercially consumed water sources can constitute a direct exposure route for the population. In invertebrate fauna, the land snail Cornu aspersum showed accumulation of MPs in its tissues and even higher excretion in its feces, indicating a continuous process of ingestion and elimination, as well as their potential role in trophic transfer. Similarly, in wild mammals such as the ringtail (Bassariscus sp.), the presence of MPs was recorded in all feces collected in urban and peri-urban areas, with concentrations that were notably higher in urban areas than in peri-urban areas, with fragments being the predominant form. In humans, MPs were detected in all urine samples from children and adolescents, with higher loads in children compared to adolescents, although without alterations in renal function parameters, suggesting bioaccumulation processes at an early age. Taken together, these findings confirm the widespread presence of MPs at multiple levels of environmental exposure and underscore the urgency of multidisciplinary research that delves deeper into the mechanisms of toxicity and their implications for human and ecosystem health.

CC 27. *The Challenges for Student Societies in Scientific Dissemination and Strengthening in a New Exposomal Era*

Iván Zapata
Universidad Autónoma de Tamaulipas

In a time defined by information overload for a few and misinformation for the vast majority of the population, the role of young people in science communication is crucial, enabling accurate and accessible communication. Student science societies are the cornerstone for fostering vocations and critical thinking, facing unique challenges in translating and disseminating complex concepts like the exposome, which addresses sensitive issues such as environmental justice, health inequalities, and individual versus collective social responsibility. What role should student societies play in navigating this public debate?

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CC 28. *DAAD Information Capsule*

Alejandro Cadena Ramos
Coordinador de Alumnos DAAD y programas institucionales. Alemania, México

Mr. Alejandro Cadena Ramos, Coordinator of Students and Institutional Programs for Mexico, Central America, and the Caribbean at the German Academic Exchange Service (DAAD), highlighted that Germany is one of the world's leading academic destinations thanks to the high quality of its universities, its cultural diversity, and a strong focus on professional and research practice. He explained that there are various types of institutions (traditional universities, universities of applied sciences, and art/music academies) throughout the country, most of them public and tuition-free, and that it is possible to find more than 20,000 study programs, including options taught in English. He emphasized that the DAAD has supported thousands of students and that currently about a third of Mexicans in Germany hold a scholarship from this organization. The main academic, linguistic, and financial requirements for accessing undergraduate or graduate studies were detailed, as well as the key steps in the process: researching programs and universities, contacting institutions, applying, arranging financing, and obtaining a visa. In addition, the different types of scholarships available according to level and area of interest were presented, such as summer courses, master's degrees, doctorates and short research stays.

CC 29. *Epigenetic Echoes: How Does Exposure to Chemicals Influence Health Across Generations?*

Aleksandra Buha Đorđević
University of Belgrade, Serbia

Recent research highlights that chemical mixtures frequently found in middle-income countries, such as Mexico and Serbia, in water, food, and air, can generate nonlinear and difficult-to-predict biological effects, requiring comprehensive experimental approaches. The scientific network led by Dr. Aleksandra Buha ĐorđevićΔhas promoted interdisciplinary studies aimed at understanding how environmental pollutants modify the epigenome and how these modifications contribute to disease. These studies have used techniques such as HPLC, ELISA, qPCR, and omics approaches such as global methylation, transcriptomics, and microRNA analysis to identify molecular patterns associated with chemical exposure. The findings indicate that certain metals alter the methylation of genes involved in inflammation and cell proliferation, while some pesticides induce imbalances in microRNAs related to reproductive development and metabolism. It is crucial to consider that the stages of highest risk correspond to gamete formation and early development, critical moments in epigenetic programming.

CC 30. *Oviductal Microenvironment and Epigenetics: An Approach From a Bovine Model of Embryonic Development*

E. Mariela Roldán Olarte
Universidad Nacional de Tucumán, Argentina

Dr. Eugenia Mariela Roldán Olarte's research, based on a bovine model, aims to understand how the oviductal microenvironment, influenced even by the female's diet, plays a significant role in cell signaling, the secretion of bioactive molecules such as proteins and glycosaminoglycans, and the epigenetic regulation of oviductal cells and the embryo. This microenvironment impacts fertilization and the first mitotic cycles, influencing chromatin remodeling and DNA methylation levels in the embryo. Alterations in the biochemical composition, whether due to physiological factors or exposure to bioactive molecules, can modify the epigenetic profile and embryonic viability. Furthermore, hormonal and regulatory signals from the oviduct contribute to establishing epigenetic reserves that are transmitted to offspring. This approach has direct implications for optimizing assisted reproductive technologies and preventing epigenetic disorders in embryos cultured in vitro.

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CC 31. *Lifestyle and Epigenetic Control of Oxidative Stress: miRNAs in the Era of Exposomics*

Hernán Mauricio Rivera Escobar
BIOPESA, Universidad del Tolima. Colombia

Oxidative stress is a key component in the pathophysiology of many chronic diseases, including cancer, and results from an imbalance between the generation of reactive oxygen species (ROS) and the antioxidant capacity of cells. In recent years, microRNAs (miRNAs) have been recognized as important epigenetic regulators of the cellular response to oxidative stress, as they post-transcriptionally control genes involved in proliferation, apoptosis, energy metabolism, and redox signaling. Growing evidence from tumor models, particularly melanoma, indicates that alterations in miRNA expression can favor both cellular adaptation to hostile oxidative environments and tumor progression. These miRNAs regulate major signaling pathways such as PI3K/AKT, MAPK, NRF2/KEAP1, and Wnt/Δcatenin, thereby influencing cellular plasticity, survival, and interactions with the tumor microenvironment. Within the framework of exposomics, lifestyle-related environmental factors—including diet, physical activity, sleep quality, stress management, and sexual activity—play a relevant role in modulating redox homeostasis and, consequently, miRNA expression patterns. Diets rich in antioxidant compounds, regular exercise, and adequate rest are associated with miRNA profiles that mitigate oxidative damage, whereas unhealthy habits tend to exacerbate redox imbalance and increase the risk of neoplastic transformation.nThe integrated analysis of environmental exposures, epigenetic mechanisms, and lifestyle factors provides valuable insights for identifying miRNA signatures with diagnostic, prognostic, and therapeutic potential in melanoma and other cancers. Overall, these findings support the development of preventive and personalized health strategies, consistent with the goals of translational medicine and modern molecular epidemiology.

CC 32. *Male Factor Infertility: Impact of Epigenetics and Exposure to Toxins on Sperm*

Jaime Paz Ávila
Universidad Autónoma de Tamaulipas. México

Male factor infertility depends not only on sperm count but also on sperm integrity and genetic and epigenetic factors. Factors such as varicocele, hormonal imbalances, and exposure to environmental toxins (pesticides, tobacco, heavy metals) generate oxidative stress and damage to sperm DNA, directly affecting reproductive capacity. This talk will address the role of DNA fragmentation, the impact of toxins, modern sperm selection techniques (IMSI, PICSI, microfluidics), and current strategies for optimizing male fertility.

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CD 1. *Genomics Applications in Epidemiological Surveillance*

Eduardo Canek Reynoso
National Institute of Public Health. Mexico. Mexico

By applying genomics to the monitoring of microorganisms in food, Dr. Reynoso and his team have detected new and emerging threats (increasing serotypes, the emergence of multidrug resistance) before they translate into large human outbreaks. Knowledge of the genetic structure of circulating pathogens in Mexico facilitates the design of better control strategies, as well as public food policies and surveillance. The systematic integration of geographic location and genomic data promotes more localized and contextualized surveillance (e.g., by meat type, region, serotype) than traditional surveillance (isolation and phenotypic resistance), as well as the use of genomic sequencing of pathogens (resistance genes, pangenome, phylogenetic relationships) for active surveillance, to identify resistance genes, and to map geographic distribution and potential transmission routes.

CD 2. *Biomarkers of Suicidal Behavior: Transcriptomic Applications*

José Miguel Chin Chan
Autonomous University of Campeche. México

According to the World Health Organization (WHO), approximately 720,000 people die by suicide each year. Young people are particularly vulnerable, as suicide is the second leading cause of death among those aged 19 to 25 (WHO, 2025). In Mexico, the Yucatán Peninsula is considered a priority region due to its high suicide rates across its states; however, biological research on this topic remains limited (INEGI, 2025). Furthermore, there are currently no validated biomarkers for suicide risk; assessments are based solely on psychiatric interviews. To address this national and regional health challenge, the IXTAB Project was established in 2022 through a multidisciplinary and interinstitutional collaboration. Its primary objective is to identify candidate molecules in the blood of patients with suicidal behavior that could serve as biomarkers for suicide risk. Patients were recruited from the Campeche Psychiatric Hospital, and blood samples were collected. Serum levels of serotonin, cytokines, and candidate microRNAs were measured using HPLC, ELISA, and qPCR, respectively. Their results showed no significant changes in cytokine levels, but patients with suicidal behavior exhibited lower serotonin levels. Several candidate microRNAs also showed differential expression. A transcriptomic approach allowed us to identify nearly 23 microRNAs with differential expression in individuals with suicidal behavior. Omics technologies are essential for identifying molecular alterations associated with mental disorders, including suicidal behavior. Therefore, further omics-based research is needed to discover reliable biomarkers and potential drug targets. A multidisciplinary approach is also crucial to achieve a significant social impact on local populations facing mental health challenges such as depression, anxiety, and suicide risk.

CD 3. Epidemiology, Statistics, and Modelling in Health and Environmental Studies

Katherine Ogurtsova
Universitätsklinikum Düsseldorf. Germany

The surveillance, characterization, and analysis of risk factors for chronic diseases, especially diabetes and its impact on cognitive function in adults, has combined population data with advanced statistical modeling. Studies include the global estimation of undiagnosed diabetes, identifying regions with the highest burden and highlighting the need to improve early diagnosis and surveillance in low- and middle-income countries, as well as the exploration of environmental and socioeconomic determinants of cognitive function. In cohort studies using population data led by Dr. Ogurtsova, the interaction between exposure to environmental factors (such as air pollution and traffic noise) and neighborhood socioeconomic conditions to influence cognitive function was analyzed. Using dimensionality reduction techniques (such as self-organizing maps) and principal component analysis, "exposure clusters" reflecting unfavorable residential environments were identified. These data allowed researchers to conclude that both greater environmental burdens and worse socioeconomic indicators in the neighborhood are associated with poorer cognitive performance, and that the combined effect was stronger than that of either factor individually—a highly relevant concept in Human Exposome consortia. Overall, the work integrates traditional epidemiological methods and complex data analysis, promoting evidence-based public health interventions and preventive policies that address the biological, environmental, and social determinants of health.

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CD 4. *Laboratory Methods and Technological Instrumentation Platforms With Artificial Intelligence*

Eduardo Coello
ORBEM. Germany

Dr. Coello's research represents a significant advancement in the field of Artificial Intelligence. By optimizing innovations in the acquisition, reconstruction, and quantification of Magnetic Resonance Imaging (MRS, especially 7T) in the medical field, studies can detect low-concentration metabolites with high sensitivity. This allows for the examination of clinical contexts such as brain and breast tumors, the effects of long COVID, traumatic brain injuries, psychiatric disorders, and neuroinflammatory conditions, among others. These technologies are helping to bridge the gap between physics, image engineering, and applied medicine by improving spatial and temporal resolution. For example, neurometabolite quantification is implemented within a framework based on random forest regression to estimate spectral parameters in MRS, trained on simulated in-vivo spectra. His training at top-level institutions (TUM, GE Research, Munich; BWH-Harvard, Boston) and his interdisciplinary approach (engineering, medical physics, clinical radiology) allow him to operate at the intersection of multiple fields, which is strategic for innovation in his field by incorporating artificial intelligence analysis for the reconstruction of spectroscopic data, data processing and development of automatic quantification algorithms.

CD 5. *R Programming With Artificial Intelligence*

Israel Aguilar Ordóñez
ITESM. México

Dr. Aguilar led a workshop on R programming for omics data analysis, which aligns with the objectives of implementing artificial intelligence in the study of the Human Exposome. During the session, "Search, extraction, mining, integration, and visualization of OMICs data," data mining and data integration techniques common in artificial intelligence applied to genomic analysis were discussed. With the strategies taught, participants are able to manage complex OMICs data comprehensively: from search to analysis and visualization, incorporating concepts from programming, statistics, machine learning, and bioinformatics. This knowledge enables the production of clean, integrated, and structured data for training predictive models, which can then be made openly available for reproducibility.

CD 6. *Omics Applications and Their Contributions to the Study of Human Diseases*

Guillermo Barreto
EPIGLYCAN, IMoPA, Université de Lorraine. France

Dr. Barreto explained how the tyrosine kinase inhibitor erlotinib (clinically used in various diseases, including EGFR-mutated non-small cell lung carcinoma (NSCLC)) can induce rearrangements in the three-dimensional (3D) organization of the genome. Using an integrative multi-omics approach, he demonstrated that erlotinib promotes the rearrangement of chromatin loops and interaction domains between enhancers and promoters, favoring the activation of tumor suppressor genes through epromoters (promoters that also act as enhancers) linked to the FOXA2 binding protein. This represents a novel epigenetic mechanism for the action of erlotinib beyond conventional EGFR inhibition, implying that the treatment modifies nuclear architecture and can also reactivate previously silenced tumor suppression pathways.

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CD 7. Emerging Exposomic Topics at Global and National Levels: Clinical Trials, Cohorts, and Epidemiology

Karla Rubio
EPIGEN-BUAP. México | MGH-Harvard. USA

Exposomics explores how the complex interaction of environmental factors, from water, air, and food pollutants to social and psychological stressors, shapes our biology. In presenting her research, Dr. Rubio integrated air pollution, atmospheric pollution (particulate matter), obesity, environmental toxicology, and epigenetic alterations within the context of exposomal clouds. These clouds are enriched by molecular signatures that link environmental exposures (e.g., cadmium, PM2.5, mercury, lithium, glyphosate, volcanic ash) with epigenomic changes (e.g., DNA methylation, chromatin structure, transcriptional horizon, liquid-liquid phase separation molecular biocondensates) and altered metabolic pathways associated with chronic diseases. For example, their bioinformatics studies with experimental validations by molecular biology and sequencing techniques have generated molecular signatures of lipogenesis, subnuclear response to Cadmium, nucleolar stress, resistance to radiotherapy, among others, and their causal relationship with oxidative stress pathways, mitochondrial and epigenomic damage, corroborating machine learning studies of exposure to atmospheric pollutants with epigenetic modifications that could favor a pathological phenotype in various contexts.

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SP 1. *The Atmospheric Pollutant Benzo[ghi]perylene Induces Epithelial–Mesenchymal Transition (EMT) in Human Bronchial NL-20 Cells In Vitro*

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Air pollution, particularly from PM2.5 particulate matter, poses a risk to respiratory health. Benzo[ghi]perylene (B[ghi]p) is the most abundant polycyclic aromatic hydrocarbon (PAH) in the atmosphere of Mexico City. Although not classified as a carcinogen, previous studies demonstrate its genotoxic potential and its ability to activate pro-inflammatory signaling pathways and cellular dormancy. It is unknown whether B[ghi]p, an abundant pollutant in the atmosphere of Mexico City, can induce epithelial-mesenchymal transition (EMT), a key process in the development of fibrosis, cancer, and other diseases. Exposure to B[ghi]p was shown to induce EMT in the human bronchial cell line NL-20. NL-20 cells were exposed to B[ghi]p (1.24 µg/mL) for 24 h. The expression of the genes SOX2, OCT4, NANOG, and ALDH1, as well as proteins (by immunocytochemistry) such as E-cadherin, N-cadherin, and Vimentin, was quantified. Morphological changes (Giemsa staining) and intercellular adhesion capacity (hanging droplet organoid formation assay) were analyzed. Exposure to B[ghi]p significantly increased the expression of the genes SOX2, OCT4, and NANOG ($p<0.05$). Increased expression of the mesenchymal and pluripotent proteins N-cadherin and vimentin was observed, along with decreased expression of E-cadherin. Morphologically, the cells lost polarity and showed a reduced capacity for spheroid formation, confirming a loss of intercellular adhesion. It can be concluded that B[ghi]p induces TEM in bronchial cells, revealing a novel toxicity mechanism with potential implications in chronic respiratory diseases and cancer.

SP 2. *Epidemiological Analysis of the Relationship Between $PM_{2.5}$ Air Pollutants and Preterm Births at the Hospital de la Mujer, 2020–2025*

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Air pollution is a global public health problem. Among pollutants, fine particulate matter (PM2.5) stands out due to its small size and chemical composition, which facilitate its entry into the human body and the generation of adverse effects. In the perinatal context, preterm births (<37 weeks of gestation) represent one of the leading causes of infant morbidity and mortality worldwide. In Mexico, the estimated prevalence is 8 per 100 births, constituting a challenge in maternal and child health. Recent studies have linked maternal exposure to PM2.5 with adverse perinatal outcomes, identifying the third trimester as a critical period of vulnerability due to mechanisms such as systemic inflammation, oxidative stress, placental dysfunction, and epigenetic alterations. To analyze the relationship between maternal exposure to PM2.5 and the occurrence of preterm births in women attended at the Hospital de la Mujer Puebla from 2020 to 2025. Observational and analytical study with a historical cohort and mixed ecological approach. Data will be collected from clinical records of births occurring between 2020 and 2025 at the Hospital de la Mujer Puebla, combined with atmospheric pollutant and environmental exposure data provided by the State Atmospheric Monitoring Network (REMA). Inclusion and exclusion factors such as maternal age and obstetric history will be controlled.

SP 3. *Systematic Study of Maternal Behavior in SHR Rats: An Experimental Model of ADHD and Hypertension*

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Maternal behavior (MB) encompasses the behaviors that the mother exhibits before and after birth, being crucial for the survival and development of the offspring. When MB is inadequate, deficient, or negligent, it can negatively impact the neurodevelopment of the progeny. Several animal models exist to study this situation, but most only present deficiencies or neglect in MB. Pilot studies of MB in SHR rats, a model of spontaneous arterial hypertension and At-

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tention Deficit Hyperactivity Disorder, show deficient and negligent MB. Therefore, in the present work we conducted a systematic study of MB in SHR rats (F0) and their normotensive control (Wistar) during postpartum days (PPD) 2, 4, 6, and 8. Results from F0 mothers showed that SHR rats displayed a significantly higher frequency of sniffing, body licking, anogenital licking, and hovering over the pups compared to Wistar mothers. In contrast, the frequency and duration of high-intensity nursing, as well as nest building, were significantly lower in SHR rats than in the control group. Additionally, SHR mothers exhibited atypical behaviors such as atypical carrying and re-carrying of pups, "hesitation," and "wandering" with the pups. Increased self-grooming and exploration were also observed. These findings suggest that the SHR rat model reflects the complexity of deficient and negligent maternal behavior, similar to that observed in humans. Consequently, this model could be valuable for investigating deficient and negligent MB, as well as its effects on progeny neurodevelopment. In fact, the daughters (F1) of both groups are currently being evaluated regarding MB, as well as certain emotional and cognitive processes.

SP 4. *Mechanistic Evaluation of Riolozatriona Against Herpes Simplex Virus Type 2*

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Herpes simplex virus type 2 (HSV-2) is a leading cause of genital and neonatal herpes and establishes latency in sensory ganglia. The standard treatment, acyclovir (ACV), inhibits the viral DNA polymerase but does not eradicate the virus, and resistant strains have emerged, warranting exploration of alternative therapies. In this context, plant-derived antivirals are of interest. Jatropha dioica and its metabolite Riolozatriona have shown anti-herpetic activity. To determine the mechanism of action of Riolozatriona against HSV-2. Virucidal assay. PBS–HSV-2 suspensions were incubated with Riolozatriona (300, 150, or 75 µg/mL) for 1 h at 4 °C and then used to inoculate Vero cells in 24-well plates. Percent plaque reduction was calculated relative to an HSV-2 infection control. Protection of Vero cells. Vero cells were pretreated with Riolozatriona at the indicated concentrations for varying exposure times and subsequently infected with HSV-2. Percent reduction in plaque formation was quantified relative to the infection control. Seeding 1.2 × 105 cells/well was required to achieve 90–95% confluence. An HSV-2 stock was prepared; plaque assay titration showed that 100 µL of the 10–3 dilution yielded 25 plaque-forming units (PFU), corresponding to 2.5 × 105 PFU/mL. Riolozatriona displayed no virucidal activity in the suspension assay and no protective effect in pretreated Vero cells under the conditions tested.

SP 5. *Green Synthesis, Characterization and Evaluation of the Antibacterial Activity of AgNPs Synthesized From Ganoderma applanatum*

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The increasing prevalence of microbial diseases, coupled with antibiotic resistance, has become a significant public health concern, leading to the exploration of alternative treatments that utilise nanotechnology. Silver nanoparticles (AgNPs) have been widely studied due to their useful physicochemical properties in various fields, including medicine. Biological synthesis, also known as 'green synthesis', offers several advantages over physicochemical methods, including environmental safety, reduced toxicity and lower energy and resource consumption. In this study, silver nanoparticles were synthesised using Ganoderma applanatum, a medicinal mushroom belonging to the Ganodermataceae family, as a reducing agent. This basidiomycete is widely recognised for its bioactive compounds, including polysaccharides, triterpenoids and phenolic compounds, which exhibit antimicrobial, antioxidant and anti-inflammatory properties. These compounds facilitate the reduction and stabilisation of silver ions during nanoparticle synthesis. The AgNPs were synthesised from Ganoderma applanatum fruiting body extracts and characterised using UV-Vis spectroscopy, obtaining a response within the expected range for silver nanoparticles. Scanning electron microscopy (SEM-EDS) and dynamic light scattering (DLS) analyses confirmed the formation and distribution of the AgNPs. UV-Vis spectroscopy revealed an absorption peak within the 420–450 nm range, confirming the formation of AgNPs. SEM-EDS identified the presence of silver in the sample. DLS analyses determined the average size and distribution of the nanoparticles. Regarding antimicrobial activity, Kirby-Bauer disk diffusion assays demonstrated significant inhibition of Staphylococcus aureus and Escherichia coli growth, indicating the antibacterial potential of AgNPs. These results demonstrate the potential of AgNPs as an alternative treatment for bacterial infections and help combat the growing problem of antibiotic resistance.

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SP 6. *Methylation of REα–eNOS Pathway Genes Associated With Maternal Pregestational Obesity in Placental and Umbilical Cord Cells*

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Introduction: *Fetal programming suggests that developmental alterations predispose individuals to metabolic and cardiovascular diseases in adulthood. In the placenta, nitric oxide (NO) promotes angiogenesis and signaling for endothelial cell proliferation and migration; estrogen receptor alpha enhances vasodilation and eNOS synthesis, thereby increasing NO in response to hormonal changes during pregnancy. The non-canonical signaling pathway and its proteins have been associated with adult diseases (e.g., cardiovascular disease and cancer), and their genes are sensitive to epigenetic modifications.* ***Methods:*** *A total of 134 samples (98 placenta and 40 umbilical cord) were analyzed from three datasets (GSE192812, GSE144129, and GSE153564). Differential methylation analysis was performed with the limma (3.64.3) and minfi (1.54.1). Enrichment analysis was conducted with clusterProfiler (4.16.0). The linear model incorporated a three-way interaction (Tissue type * Maternal BMI classification * Neonatal sex).* ***Results:*** *Our findings suggest sex-dependent epigenetic programming in umbilical cord genes: in females CALM1-3, ITPR1, PIK3CD, NOS3; in males AKT2, PIK3R2, SRC; and in both sexes ITPR3 and PLCB3. In the placenta, no differential methylation signal was detected in the pathway, which may indicate a tissue-specific effect of pregestational obesity. Enrichment analysis revealed alterations in males involving endocytosis, PRC, and chromatin remodeling (linked to epigenetic reprogramming and intracellular trafficking), whereas in females angiogenesis (Notch), Rap1, AMPK, and autophagy pathways were affected, supporting a greater activation of NOS3.* ***Conclusion:*** *Pregestational obesity is associated with sex-specific methylation changes in the REα-eNOS pathway of the umbilical cord, with a stronger effect in female neonates, along with alterations in related interconnected pathways.*

SP 7. *Prevalence of Gastrointestinal Medicine Use in Children: An Analysis of NHANES 1999–2018*

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Background: *Functional gastrointestinal disorders (FGIDs) have increased in recent years, with a prevalence ranging from 12% to 29% in children and adolescents. This trend may influence the use of medications prescribed to treat these conditions.* ***Problem statement:*** *There is limited evidence on the actual prevalence of gastrointestinal medication use in this age group. This gap complicates the identification of issues such as polypharmacy or inappropriate prescribing, as well as the understanding of associated factors such as age, ethnicity, and temporal trends.* ***Objective:*** *To identify the prevalence of gastrointestinal medication use among adolescents.* ***Methods:*** *Data were obtained from the National Health and Nutrition Examination Survey (NHANES), covering the 1999–2018 cycles. Adolescents aged 12 to 18 years, with or without a medical prescription for FGIDs, were included. The Chi-square test was used to analyse the prevalence of gastrointestinal medication use by ethnicity, sex, and survey cycle.* ***Results:*** *The cohort included 8,145 adolescents aged 12–18 years (weighted n=17,508,765). Overall, 13.27% reported using gastrointestinal medications. Prevalence was higher among females (1.9%) compared to males (0.7%, p=0.026). Non-Hispanic white adolescents showed a higher prevalence (1.8%) compared with Mexican American adolescents (0.7%, p=0.030). An increase in prevalence was observed between 1999 (3.1%) and 2018 (9.2%), although the difference was not statistically significant (p=0.338).* ***Conclusions:*** *Biological sex and ethnicity are significant determinants of gastrointestinal medication use among adolescents. Further research is needed to better understand the factors driving these disparities and their impact on clinical management and public health.*

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SP 8. *Exposome Analysis in Patients From the Instituto Nacional de Cancerología (INCan) Mexico Who Develop Oral Squamous Cell Carcinoma (OSCC)*

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The term "exposome", refers to all those environmental factors to whom the human being is exposed to along their life, according to 3 environments: general external, specific external and the internal. Analyzing exposome allows the identification of biomarkers to predict actual or past exposures or the detection of early preclinical conditions allowing early prevention strategies. Oral Squamous Cell Carcinoma (OSCC) is one of the most common affections in head and neck cancers developed in the epithelium. Analysis of the predisposing conditions in patients from the Instituto Nacional de Cancerologia (INCan) *that developed OSCC. 24 patients who assisted consultation at the department of Head and Neck oncology department at INCan were analyzed with a form application, to know their personal life conditions. 13 men and 11 women. From the 24 patients analyzed, 2 women and 2 men presented chronic systemic illness, mainly diabetes and high blood pressure. Respecting the cancer family history, 17% of men and 25% of women referred to present cases in their direct family. 37.5% presented a lesion evolution in a time under 6 months, 29% over 6 months and 33.5% an evolution bigger than 1 year. The most harmful personal health habits in both groups were: overweight or obesity, smoking, alcoholism, consumption of processed food and high intake of sugared beverages. Finally, both groups manifested tooth brushing as their most useful practice for dental hygiene; furthermore, only a third of the patients perceive their oral hygiene as "acceptable". It is necessary to promote higher quality preventive measures in oral hygiene among the population to optimize timely detection.*

SP 9. *Alterations in the Cytoarchitecture and Neuronal and Glial Activity of the Prefrontal Cortex in a Model of Depression*

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Major depressive disorder (MDD) is a prevalent psychiatric illness worldwide. One of its clinical subtypes is agitated depression, characterized by the coexistence of affective symptoms and psychomotor agitation. The latter typically presents as increased motor activity, including hyperlocomotion and repetitive, purposeless movements. Due to the severity of their symptoms, specific management recommendations should be developed and new therapeutic options considered. Olfactory bulbectomy (OBX) is one of the most representative animal models for studying depression with agitated features. OBX involves bilateral ablation of the olfactory bulbs, which causes changes in the endocrine, immune, and neurotransmitter systems similar to those observed in patients with MDD. The open field test (OFT) is commonly used in rodents to measure exploratory behavior and rearing or grooming frequency, in order to predict their emotional state. OBX rodents exposed to OFT show hyperlocomotion and repetitive behaviors. We used the prefrontal cortex (PFC) of 75 adult male Wistar rats (Rattus norvegicus) with OBX to determine the influence of astrocytes, microglia, pyramidal neurons, nitric oxide (NO), and neuronal activity on the behavior observed in the OFT. We found that OBX generated hyperlocomotion and increased rearing frequency. Furthermore, it increased the number of cells labeled with glial fibrillary acidic protein (an astrocyte marker), c-Fos (a marker of neuronal activity), and NO levels, while decreasing the density of dendritic spines in pyramidal neurons in the PFC. In conclusion, OBX induces agitated behavior, mediated by modifications in astrocytes of the PFC, which trigger oxidative stress linked to neuronal activation, along with other neurobiological mechanisms. Our findings should be considered to optimize financial resources for the evaluation of potential antidepressant, anxiolytic, and neuroprotective drugs.

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SP 10. *Visceral Adiposity as a Key Predictor of Non-Alcoholic Fatty Liver Disease in Adolescents: An Analysis of NHANES 2017–2018*

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Obesity, particularly visceral adiposity, has increased to 21% in adolescents. This is a key risk factor for the development of nonalcoholic fatty liver disease (NAFLD). Therefore, the objective of this study was to identify the predictive risk value of visceral adipose tissue (VAT), subcutaneous adipose tissue (SAT), and total fat (TAFA) and their association with NAFLD in adolescents. ***Methods:*** *The 2017-2018 NHANES database was used. Using inclusion and exclusion criteria, a sample of 610 adolescents (12-17 years) was formed. NAFLD was determined by CAP-USG (controlled attenuation parameter ultrasound). Demographic, anthropometric, and body composition variables were analyzed using linear and multivariate logistic regressions and ROC curves to determine the association and predictive capacity between VAT, SAT, TAFA, and NAFLD, adjusting for age, sex, and ethnicity.* ***Results:*** *45.9% of the sample had NAFLD, with a higher prevalence in adolescents of Mexican ethnicity (p=0.022). NAFLD was associated with increased BMI, waist circumference, and abdominal fat measurements (p<0.001, p<0.001, p<0.001, respectively). VAT showed a higher correlation with USG-CAP (r=0.558, p<0.001) compared to SAT (r=0.502, p<0.001) and TAFA (r=0.529, p<0.001). In multivariate analyses, VAT maintained the most robust association. VAT also showed the greatest predictive power for NAFLD (AUC=0.77), with an optimal cutoff of 49 cm² (sensitivity=59%, specificity=83%). Both SAT and TAFA showed slightly lower predictive values.* ***Conclusions:*** *Visceral adiposity was not only significantly associated with NAFLD in adolescents but also proved to be the best predictor of NAFLD compared with other abdominal fat depots. These findings underscore the importance of evaluating VAT in pediatric populations as a useful tool for the early detection of NAFLD.*

SP 11. *Fetal Epigenetic Reprogramming Mediated by the Intestinal Microbiota: A Key Axis in the Origins of Obesity and Diabetes*

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SP 12. *Retroperitoneal CIC-DUX4 Sarcoma: The Role of Next-Generation Sequencing in Diagnostic Reclassification*

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Synovial sarcoma (SS) is a malignant mesenchymal tumor that accounts for 5-10% of all soft tissue sarcomas (STS). Its diagnosis is often challenging due to considerable overlap in clinical, radiological and histopathological features with other STS, leading to a substantial risk of misdiagnosis. Advanced molecular testing, such as next generation sequencing (NGS), plays a pivotal role in differentiating rare entities, including CIC-DUX4 sarcoma, characterized by an aggressive clinical course and poor prognosis. We present the case of a 38-year-old female with a left retroperitoneal mass. Initial histopathological evaluation following a radical nephrectomy suggested SS (TLE1, BLC2 +). However, NGS identified a CIC-DUX4 fusion, resulting in a revised diagnosis of CIC-DUX4 sarcoma (CDS). The disease course was marked by multiple recurrences and metastasis managed with surgery, radiotherapy and doxorrubicin-ifosfamide chemotherapy. Additionally, the molecular profile revealed an FGFR3 R248C mutation, an unusual finding in this context. Despite the tumor's aggressive behavior, the patient achieved an overall survival of 36 months, exceeding the reported median survival of 15.4 months. This case illustrates the diagnostic challenges associated with CDS and underscores the essential role of incorporating advanced molecular analyses into patient assessment. CDS is a rare, highly aggressive sarcoma often resistant to conventional chemotherapy, emphasizing the need for novel targeted therapies. Molecular profiling is essential for the accurate diagnosis and management of rare sarcomas like CDS, enabling identification of therapeutic targets and guiding personalized treatment.

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SP 13. *Role of Serum α-Klotho in Patients With Metabolic Syndrome With and Without Insulin Resistance*

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Background: *The α-Klotho protein is an anti-aging peptide involved in metabolic regulation. Although an association between its serum concentrations and various metabolic pathologies like metabolic syndrome (MetS) and insulin resistance (IR) has been described, the combined relationship of these conditions with Klotho levels has not been explored.* ***Objective:*** *To determine the association between serum α-Klotho levels and IR in patients with and without MetS.* ***Methods:*** *A cross-sectional study was conducted using data from 3,842 participants from the NHANES data base (2007-2016). Subjects were categorized by the presence of MetS and IR. Klotho concentrations were divided into quartiles. The association was assessed using Pearson correlation, IR risk was estimated with logistic regression (OR, 95% CI), and variable interaction was analyzed with a generalized linear model (mean ± standard error). A p-value <0.05 was considered significant.* ***Results:*** *A positive linear correlation was found between Klotho levels and HOMA-IR (r=0.035, p<0.001). In the MetS group, the lowest Klotho concentrations showed a protective effect against IR (Q1: OR=0.450, 95% CI 0.269–0.754, p=0.003). Klotho levels were significantly higher in the MetS+/IR– subgroup (865.17±12.46) compared to the other groups (p<0.001).* ***Conclusion:*** *A direct association exists between serum Klotho levels and IR in the context of MetS. Lower serum Klotho concentrations may confer protection against the development of IR in patients who already have metabolic syndrome.*

SP 14. *Molecular Bases for the Design of a Hypoxia Biosensor*

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Girardinichthys multiradiatus is an endemic and vulnerable species from the Cuenca Alta del Rio Lerma (Estado de México) *that develops under aquatic hypoxia conditions aggravated by factors such as pollution, poor water resource management, and low barometric pressure. The species' ability to survive under such conditions has sparked growing interest in studying its mechanisms of adaptation to hypoxia, particularly the pathway regulated by Hypoxia-Inducible Factor 1 (HIF-1). HIF-1 is composed of an oxygen-sensitive subunit (HIF-1α) and a constitutively expressed subunit (ARNT). This complex functions as a molecular regulator under hypoxic conditions, activating the transcription of numerous genes, including those involved in energy metabolism, angiogenesis, apoptosis, and other genes whose products promote cell survival under hypoxia. This work aims to comprehensively analyze the structural, functional, and topological properties of the HIF-1 complex in G. multiradiatus through molecular modeling, docking with hypoxia response elements (HREs), and network theory, in order to identify the most relevant domains and residues in its adaptation mechanism to hypoxic environments and their potential application in the design of a hypoxia biosensor. To achieve this, the three-dimensional structures of HIF-1α and ARNT were built, and the contact network of the basic Helix-Loop-Helix (bHLH) domain in HIF-1α was generated, evaluating the centrality measures of its residues. Preliminary results show high connectivity and centrality in specific regions of the bHLH domain, suggesting a conserved functional core. These findings could provide a solid structural basis for future stages of complex analysis, opening the possibility of developing effective strategies for the implementation of a hypoxia-sensitive biosensor with applications in biomedicine and biotechnology.*

SP 15. *Fiber Optic Sensor Based on Evanescent Waves for the Measurement and Determination of Optical Parameters in Cell Culture Media*

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Optical fibers work by the physical phenomenon of total internal reflection, where an electromagnetic wave strikes the interface between a denser medium and a less dense one. Although the energy is not transmitted to the less dense medium, it generates an evanescent field that decays exponentially with distance from the interface. Evanescent wave-based fiber optic sensors are photonic devices used for the detection and quantification of physicochemical changes.

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This paper reports on an evanescent wave-based fiber optic sensor for the functional study of human cells in culture. The sensor was manufactured by removing the coating from a section of optical fiber using hydrofluoric acid (HF). The etching lasted 1 hour, exposing the fiber core to a diameter of approximately 6 µm. The optical transmission percentage and etching time were evaluated, observing a decrease in transmission as the time increased. The etching rate of 1.68 µm/min was estimated from experimental data. The sensor detected culture medium (10% fetal bovine serum and 1% penicillin/streptomycin) and MDA-MB-231 breast cancer cell supernatants. Optical parameters such as refractive index (n), evanescent field penetration depth (D_p), and light propagation velocity in the medium (v) were calculated. The cell culture medium showed an optical transmission of 92%, with n=1.331, v=2.25×10^8 m/s, and D_p=2.20 µm. The supernatant exhibited n= 1.374, v=2.18×10^8 m/s, and D_p= 2.366 µm. Preliminary results show that the manufacturing method and the generation of evanescent waves allow optical variations to be related to biological media and cellular processes to be monitored non-invasively.

SP 16. *Molecular Biomarkers in Immunotherapy: PD-L1, TMB, and MSI as Predictors of Pembrolizumab Response in Mexican Patients With Solid Tumors*

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Pembrolizumab has demonstrated clinical benefit across multiple solid tumors; however, real-world evidence from Mexico remains limited. Biomarkers such as PD-L1 expression, tumor mutational burden (TMB), and microsatellite instability (MSI) are used to guide immunotherapy, although their predictive value may vary across populations. This study aimed to evaluate progression-free survival (PFS) and to describe associated clinical and molecular characteristics in Mexican patients treated with pembrolizumab. We conducted a retrospective cohort study including 59 patients with advanced solid tumors treated at a private oncology center in Mexico. Clinical, histopathological, and molecular data (PD-L1, TMB, MSI) were collected. PFS was estimated using the Kaplan–Meier method, and prognostic factors were assessed using univariate and multivariate Cox proportional hazards regression. The median age was 62.6 years; 48.3% were female, and 60.3% had comorbidities. The most frequent tumor types were lung (27.6%), endometrial (10.3%), breast, and renal cancers (8.6% each). Histologically, tumors were classified as adenocarcinoma (44.8%), squamous cell carcinoma (43.1%), and other histologies (12.1%). PD-L1 expression was available in 44.8% of cases, TMB in 29.3%, and MSI status in 17.2%. Median PFS was 17.1 months (95% CI: 12.9–32.5), with an objective response rate of 12.06%. Tumors with non-adenocarcinoma and non-squamous histology showed significantly longer PFS (42.0 months; HR: 0.22, p = 0.031). No significant differences in PFS were observed according to MSI status (MSI vs microsatellite stable; HR: 0.45, p = 0.311) or TMB (high vs low; HR: 1.66, p = 0.402). Only numerical differences were observed according to PD-L1 expression (>10%: 21.9 months; 1–10%: 16.3 months; 0%: 8.89 months). In this real-world Mexican cohort, histological subtype was the only factor significantly associated with PFS, while PD-L1, TMB, and MSI did not demonstrate predictive value. Nevertheless, durable responses were observed, with PFS rates of 39.4% at 24 months, 26.3% at 36 months, and 8.2% at 48 months. These findings highlight the need for novel biomarkers and for generating region-specific evidence on immunotherapy in Latin America.

SP 17. *Relationship Between Symptomatology and Probable Presence of Intestinal Protozoa in Schoolchildren From a Highly Marginalized Neighborhood in Veracruz*

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Intestinal parasitoses represent a global public health problem, particularly in communities with unfavorable socioeconomic conditions. The most frequent protozoa include Entamoeba histolytica/dispar, Giardia lamblia, and Endolimax nana, whose infections are associated with gastrointestinal symptoms such as diarrhea, abdominal pain, nausea, and vomiting. Evidence suggests that the prevalence in children from marginalized areas exceeds the national average, reflecting environmental determinants, hygiene habits, and limited access to health services. Studies in Latin America highlight that these infections affect physical and intellectual development and require timely preventive and diagnostic interventions.
In highly marginalized communities in Veracruz, there is limited information regarding the relationship between clinical symptomatology and the presence of intestinal protozoa in schoolchildren. The lack of early diagnosis and medical

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follow-up can lead to malnutrition, growth retardation, and poor academic performance, underscoring the need for studies that identify reliable clinical indicators to guide public health interventions. To evaluate the incidence of protozoan parasitosis and its correlation with gastrointestinal symptoms in schoolchildren aged 4 to 7 years, with the aim of generating evidence to support early diagnostic strategies and public health interventions. A quantitative, cross-sectional, analytical, descriptive, and exploratory study was conducted with 31 schoolchildren. A structured anamnesis was applied to record symptoms such as nausea, vomiting, abdominal pain, diarrhea, dizziness, and headache. Serial stool samples (three per child) were collected for coproparasitological analysis, using flotation and direct coproparasitological techniques. Data were analyzed through descriptive statistics, Fisher's exact test, and Cronbach's alpha (α = 0.79) to assess internal consistency. Of the schoolchildren, 43.75% were female and 56.25% male. The probable incidence of intestinal protozoa was estimated at 58.06%. Nausea (p = 0.0251), vomiting (p = 0.0107), and hypotension (p = 0.029) were significantly associated with parasitic infection. Furthermore, 41.93% did not complete the study, highlighting the need for follow-up strategies. These findings suggest that specific symptomatology may serve as an early indicator of parasitosis in vulnerable communities, emphasizing the importance of timely diagnosis, a multidisciplinary approach, and public health prevention policies.

SP 18. *Comparative Evaluation of MoCA and TEPSI Genetic Tests to Identify Psychomotor and Neurocognitive Development in Pediatrics: Pilot Test*

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The Intelligence Quotient (IQ) is a standardized measure of cognitive abilities. Neurocognitive alterations in pediatric populations may originate from genetic or metabolic causes. In Mexico, there are no cognitive assessment campaigns, which are essential to identify developmental delays requiring medical, therapeutic, or educational intervention. In the USA, the Montreal Cognitive Assessment (MoCA) test is routinely used. IQ can be assessed through the Psychomotor Development Test (TEPSI). The MoCA focuses on higher cognitive functions, leaving aside psychomotor and language aspects evaluated by TEPSI. This could limit its capacity for comprehensive detection in pediatric stages. Assessment of psychomotor and neurocognitive development in pediatric patients using the MoCA and TEPSI tests.
-Methodology: Observational, descriptive, and comparative study. Records from the Genetics Department, FACMED-BUAP (2018–2025), were reviewed. Pediatric patients aged 2 to 11 years, healthy, were included. Parents signed informed consent, ensuring participants' confidentiality. Somatometry (height, arm span, upper/lower segment, and Waardenburg index) was performed using standardized photographs. TEPSI and MoCA were applied individually. Statistical analysis was performed with SPSS v.26. A total of 148 records with complete TEPSI and 35 with both tests were included. 51% were female and 49% male. Somatometry showed that 14.6% were symmetric, while 85.4% presented some asymmetry. The mean score was 63±8.23 in TEPSI and 22.5±6.2 in MoCA. The correlation between both tests was low (r=0.097), being higher in the TEPSI motor subtest with MoCA (R^2=0.135). We therefore conclude that MoCA does not adequately assess coordination and language areas in pediatric populations. TEPSI is essential for a comprehensive evaluation of child development, and thus, both instruments are not equivalent.

SP 19. *Methylation Profiles in the NOS3 and ESR1 Genes in Familiar Trios With and Without Cardiovascular History: Association With Carotid Intima-Media Thickness in Adolescents*

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Cardiovascular diseases (CVD) are the leading cause of death, and their incidence in young people are on the rise. Intergenerational evidence suggests an epigenetic transmission of risk, where DNA methylation may function as a biomarker. Genes like NOS3 and ESR1 have shown epigenetic alterations linked to vascular dysfunction and CVD. The study's endpoint was to characterize the methylation of these three genes in family trios with and without a history of cardiovascular disease and to evaluate their relationship with carotid thickening in adolescents. Twenty-three families (63 individuals; 15 cases and 8 controls) were studied. DNA from peripheral blood was analyzed using a panel targeting 3 CpG islands in NOS3 and 8 in ESR1 (DBCAT), following the NEBNext Enzymatic Methyl-seq Illumina MiSeq protocol. The analysis included quality control, alignment, and methylation extraction, with statistical evaluation using mixed linear models by family. A total of 4.2 Gb of data were generated, with 91.3% of reads at Q30 and 100x coverage. In NOS3, islands 1 and 2 did not show significant differences, although island 2 showed a trend toward greater methylation in case families (p=0.058). Island 3 showed significant hypermethylation in cases compared to controls (p=0.02).

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In ESR1, island 4 also presented hypermethylation in case families (p=0.02), while the remaining islands did not show relevant differences.These findings suggest a differential pattern in specific regions, which reinforces the hypothesis that methylation in vascular genes contributes to the inherited susceptibility to CVD. Differential methylation in NOS3 and ESR1 could be an early risk biomarker in the descendants of patients with premature coronary disease. These epigenetic profiles, when integrated with familial and clinical factors, offer potential for preventive strategies, providing a basis for precise interventions in young populations.

SP 20. *Deficient and Neglectful Maternal Behavior in SHR Rats: Effects on Offspring Body Weight and Blood Pressure in a Cross-Fostering Study*

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Recently, we found that the maternal behavior (MB) of lactating SHR rats, a model of Attention Deficit Hyperactivity Disorder (ADHD) and spontaneous arterial hypertension is deficient and neglectful. Considering that both deficient and neglectful MB negatively affect offspring development, their combination may have a greater impact. Among the phenotypic characteristics of this model are arterial hypertension and low birth weight, alterations that persist into adulthood. Based on this, we evaluated whether the causal factors of these problems are genetic or due to deficient and neglectful maternal care (epigenetic). To assess this, we used a cross-fostering design and formed four groups: 1) SHR offspring raised by SHR mothers (SHR-SHR), 2) Wistar offspring raised by Wistar mothers (Wistar-Wistar), 3) SHR offspring raised by Wistar mothers (SHR-Wistar), and 4) Wistar offspring raised by SHR mothers (Wistar-SHR). On postnatal day (PND) 1, litters were randomly adjusted to 4 males and 4 females, and weighed (by litter) on PND 2, 4, 6, 8, and 180–210 (individual weight). We found that SHR-SHR offspring had a significantly lower body weight compared to Wistar-Wistar, SHR-Wistar, and Wistar-SHR offspring. However, in adulthood, SHR-SHR rats (females only) again weighed less than Wistar-Wistar and Wistar-SHR, but similar to SHR-Wistar. In adulthood, systolic and diastolic blood pressure of SHR-SHR rats was significantly higher compared to Wistar-Wistar, SHR-Wistar, and Wistar-SHR rats. These findings suggest that the observed changes are the result of a combination of both genetic and/or epigenetic factors.

SP 21. *Effects of a hyperglycemic diet on learning and spatial memory in first filial generation male Long Evans rats.*

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Metabolic syndrome (MS) is a multifactorial disorder characterized by obesity, insulin resistance, dyslipidemia, and hypertension, which significantly increases the risk of developing type 2 diabetes and cardiovascular diseases. Growing evidence associates MS with cognitive deficits and a higher risk of neurodegenerative diseases, such as Alzheimer's disease. The objective of this study was to evaluate the impact of a high-fructose diet, as an inducer of an MS phenotype, on spatial learning and memory in first-filial generation male rats. Adult male Long Evans rats of the first-filial generation were used, in which the MS phenotype was induced by the intake of a high-fructose diet for 3 months starting from weaning. The animals were subjected to the Novel Object Recognition Test (NORT) to evaluate short- and long-term memory. Exploratory behavior was quantified through the recognition index (IR) and the discrimination index (ID). The data were statistically analyzed using a Student's t-test. During the familiarization phase, both groups showed similar object exploration. However, in the memory tests, the group with diet-induced MS showed a significant reduction in both the IR and the ID compared to the control group in the long-term memory test ($p < 0.05$). These results support the hypothesis that systemic metabolic alterations have a detrimental effect on cognitive functions, and suggest that this murine model is suitable for the study of the neurobiological mechanisms underlying MS-associated cognitive dysfunction.

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SP 22. *Differential Expression of NF-κB Genes in MCF-7 Cells, a Luminal A Model of Breast Cancer*

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The NF-ΔB pathway regulates inflammation, proliferation, and survival in breast cancer. In luminal A, modeled by MCF-7 (ER+/PR+, HER2–), defining basal expression and response to proinflammatory stimuli is required. Recent evidence suggests NF-κB-mitochondrial interactions and differences between subtypes. In this study, we characterized the basal and differential expression of NF-κB pathway genes in MCF-7 cells after stimulation with TNF-α. MCF-7 cells were cultured in MEM + 10% fetal bovine serum to 80% confluence and treated with TNF-α (10 ng/mL, 60 min) or vehicle. RNA was isolated (TRIzol), cDNA was synthesized, and RT-PCR was performed for RELA (p65), NFKB1 (p50), MYD88, TRAF6, CREB1, NFKBIA (IkBα), IKB8 (IKKβ), and IL10. Amplicons were separated on 2% agarose and quantified by semi-quantitative densitometry normalized to GAPDH; –RT and NTC controls were included. Size was verified with a marker. The signal was quantified as integrated density, and differences were evaluated using a t-test ($p<0.05$). Three biological replicates were performed. At baseline levels, the expression of RELA, NFKB1, MYD88, TRAF6, CREB1, NFKBIA, and IKB8 was high; IL10 was undetectable. Following TNF-α, band intensity increased for RELA, NFKB1, MYD88, and TRAF6 compared to control, consistent with NF-κB activation; CREB1 and NFKBIA showed discrete variations, and IKBKB remained stable. –RT/NTC controls did not amplify, and reproducibility between replicates was confirmed. TNF-α-induced changes in sentinel genes support the utility of MCF-7 as a luminal A model for exploring inflammatory regulation. We conclude that MCF-7 exhibits a transcriptional profile consistent with NF-κB signaling and responds with differential modifications in key genes. We suggest confirming by qPCR (ΔΔCt) and validating by Western blot or phosphorylated p65 immunofluorescence. Standardization of conditions and controls strengthens the comparability of future basic studies with translational implications in luminal A breast cancer.

SP 23. *Evaluation of Serum Levels and Magnesium Intake in Patients With and Without Type 2 Diabetes*

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Type 2 Diabetes(T2D) is a frequent and silent disease that prompts research into which minerals helps into collaboration. The hyperglucemia causes cardiovascular and kidney disease. Magnesium have been little studied in patients with diabetes, analyzing it's necessary to understand the clinical impact and make prevention and nutritional treatment strategies that allow us to have a better comprehensive management of the pathology. To assess serum levels and magnesium intake in patients with and without type 2 diabetes. An observational, comparative, prospective study was designed, including 118 individuals diagnosed with T2DM and 63 without T2DM, all beneficiaries of UMF-2, IMSS-Puebla. The study population was characterized anthropometrically, metabolically, and by serum magnesium levels. Magnesium intake was assessed using a 7-day food diary. Our results show a difference in age between patients with and without T2DM (52.2 ± 11.5 vs 45.4 ± 13.3; $P<0.05$). No significant differences were observed in biological sex. However, significant differences were found in BMI between patients with and without T2DM (30.0 ± 5.7 vs 27.1 ± 4.3; $P<0.001$). Significant differences were also observed in biochemical parameters between patients with and without T2DM (FPG, HbA1c, Insulin, TG, HOMA-IR, HOMA-B, C-peptide, mainly). On the other hand, no significant differences were found in serum magnesium levels (2.22 ± 0.2 vs 2.22 ± 0.2; $P>0.05$). However, when evaluating dietary intake of this mineral, significant differences were observed between patients with and without T2DM [304.5 (259.2–360.6) vs 328.7 (284.9–385.6); $P=0.042$]. Our results show that the patients with T2D (+) patients ingest less magnesium compared to DT2(-) patients. Prospective studies are required to establish the magnesium role of magnesium in the pathophysiology of T2D.

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SP 24. *Quantification of 17β-Estradiol in Ovary-Associated Prevertebral Ganglia During the Estrous Cycle in the Zucker Rat: Comparison Between Obese and Lean Phenotypes*

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Obesity has become a major public health issue due to its role in the development of various comorbidities, especially those related to cardiovascular and endocrine disorders. While recent research has emphasized the relationship between the central nervous system and obesity, there is growing evidence that peripheral nervous system structures, such as prevertebral ganglia, also play important roles in metabolic regulation and adipose tissue homeostasis. We analyzed the presence of 17β-estradiol in five prevertebral ganglia in female Zucker rats, both lean and obese, across the estrous cycle. These ganglia are known to influence the enteric nervous system, adrenal glands, and abdominal organs, and are also anatomically and functionally linked to the ovaries and reproductive tract. Given that the ovary, a key producer of steroid hormones, may alter its hormonal output in response to changes in both central and peripheral neural signaling, we sought to examine how 17Δestradiol levels within these ganglia fluctuate during the estrous cycle. Female Zucker rats (3 months old, n = 20) were housed under controlled conditions, and their estrous cycle stages were carefully monitored. On each day of the cycle, five animals were selected for ganglia extraction. The collected tissues were preserved and processed for analysis using immunofluorescence techniques to assess 17β-estradiol immunoreactivity in neurons of the prevertebral ganglia throughout the estrous cycle. All experimental procedures were approved by the Mexican Council for the Care and Use of Laboratory Animals. Obese rats exhibited disrupted estrous cycles, characterized by prolonged diestrus I stages, whereas lean rats maintained regular cycles. In lean animals, 17β-estradiol-immuno positive neurons peaked during diestrus II and proestrus and were lowest during diestrus I. Additionally, we observed asymmetric immunoreactivity between paired suprarenal and celiac ganglia. Soma size varied significantly throughout the cycle, with the largest neurons found during proestrus and the smallest during diestrus I. In contrast, obese rats exhibited a significantly reduced number of 17β-estradiol-positive neurons across all ganglia, with no significant variation in soma size or left-right asymmetry. These findings suggest that 17β-estradiol expression in prevertebral ganglia is dynamic and estrous-cycle-dependent in lean animals but remains relatively static under obese conditions. This hormonal modulation may influence lipid metabolism and contribute to reproductive pathologies such as polycystic ovary syndrome (PCOS), endometriosis, and infertility.

SP 25. *Determination of Expression Patterns of snoRNAs in B-Cell Acute Lymphoblastic Leukemia Patients and Their Role in Leukemogenesis*

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B cell Acute Lymphoblastic Leukemia (B-ALL) is an hematological malignancy characterized by the accumulation of immature lymphoblastic cells in bone marrow, peripheral blood and other tissues. This represents up to 95% of the ALL cases registered and is the most common type of leukemia in Mexican children, being one of the most important causes of death in people younger than 29 years old. Small nucleolar ARN (snoRNA) are a type of small non-coding ARN (sncRNA) that take part in processing rRNA and mRNA essential for the ribosomal machinery and spliceosome. In different types of leukemia, as in AML and ALL, occurs a global subexpression of snoRNAs related with specific chromosomal abnormalities, like what happens in the SLK1-DIO3 locus, suggesting an important role in developing and classification of the leukemia. In addition, it has been reported an aberrant expression of SNORD109A, SNORD64, SNORD107, SNORD35b, SNORD46 and different subtypes of SNORD116 in ALL-B, however their role in leukemogenesis remain undisclosed. There is no report of expression patterns of snoRNAs in Mexican children with LLA-B that lead to an early diagnosis and a prognostic stratification. Objective: to determine expression patterns of snoRNAs in ALL-B patients and their role in leukemogenesis. Methods: total RNA was extracted from Peripheral Blood Mononuclear Cells (PBMC) of patients diagnosed with Pro B-ALL (21), Pre B-ALL (10), Pro/Pre B-ALL (9), negatives (3) and non-hematological subjects (3). Pooling sequencing was selected and pools were sent to CoreLab: Genomics (Tec de Monterrey) for library construction and RNA sequencing by Illumina NovaSeq600 (50 M reads, paired end, 100 bp). Differential expression analysis will be made with an own script (Cutadapt, STAR, featureCounts, DESeq2). snoRNAs detected will be validated with RT-qPCR in an independent cohort, correlation studies with clinical parameters (like hemogram parameters, age, flow cytometry) and cellular pathways by functional enrichment also will be executed. Results: main discoveries found in this research will be discussed in the meeting.

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SP 26. *Importance of Folic Acid Supplementation in the Prevention of Congenital Anomalies in a Social Security Population in Puebla*

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Congenital anomalies represent a heterogeneous group of structural and functional abnormalities that arise during intrauterine development. These conditions are influenced by a complex interplay of genetic and environmental factors. In 2024, a total of 3,667 cases of congenital anomalies were reported in Mexico, corresponding to an incidence rate of 179.4 per 100,000 live births. Despite the significant burden, there remains a paucity of data regarding maternal risk factors associated with these conditions. To clinically characterize mothers of children diagnosed with congenital anomalies. A descriptive, observational, cross-sectional, and ambispective study was conducted in the Genetics Department of the General Hospital of Zone No. 20, part of the Mexican Social Security Institute in Puebla. The study included 35 mothers of pediatric patients diagnosed with congenital anomalies. Data collected included maternal age, nutritional status, sociodemographic characteristics, anthropometric measurements, and environmental exposures. The mean maternal age at the time of pregnancy was 27.49 years (±5.58). The median gestational age at the time of pregnancy diagnosis was 6 weeks (interquartile range [IQR]: 4.0–12.0). Only 2 mothers (5.7%) reported initiating folic acid supplementation during the preconception period. Of the total cases, 23 (65.7%) presented with isolated congenital anomalies, while 12 (34.3%) exhibited multiple anomalies. The most prevalent anomaly was unilateral cleft lip and palate, accounting for 22% of all cases. The findings highlight a delay in the initiation of folic acid supplementation among the majority of mothers, often beyond the critical window of early embryonic development. Most anomalies identified in this cohort are folate-sensitive, with a predominance of craniofacial malformations such as cleft lip and palate. These results underscore the need to reinforce public health strategies promoting preconception folic acid supplementation and to provide timely genetic counseling to women with a history of offspring affected by congenital anomalies

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SP 27. *Epidemiological Relationship Between PM2.5 Air Pollution and Macrosomic Newborns Born at the Hospital de la Mujer Puebla, From 2020 to 2025*

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Pregnant individuals exposed to environmental PM2.5 pollution have a greater risk of adverse perinatal outcomes, such as abnormal birth weight, which could determine the newborn's long-term health conditions. Scientific evidence reports that for every 10 µg/m3 increase in PM2.5 concentration during the second trimester of pregnancy, the risk of macrosomia. Despite being a topic of current scientific relevance, epidemiological research in Mexico has not established yet the relation between the exposure to environmental pollutants and adverse perinatal outcomes. Therefore, the Puebla State Ministry of Health, and the Hospital de la Mujer Puebla, formed a multidisciplinary team to conduct the first epidemiological analysis of its kind, from which this research is derived.m To evaluate the association between exposure to PM2.5 air pollutants and the occurrence of macrosomic newborns at the Hospital de la Mujer de Puebla from 2020 to 2025. This is observational and analytical study with a historical cohort and mixed ecological approach. Individual data from the clinical records of births at the Hospital de la Mujer de Puebla between 01/01/2020 and 31/12/2025 , is being analyzed. These data will be combined with aggregated air pollutants environmental exposure data from the State Air Monitoring Network (REMA). Exclusion and inclusion factors such as maternal age, obstetric history, among others, are controlled

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SP 28. *Gestational Exposure to Polystyrene Nanoplastics Alters Uterine Histomorphology in F0 Rats*

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The ubiquity of polystyrene nanoplastics (PS-NPs) has raised increasing concern regarding their health effects. These PS-NPs can cross biological barriers and accumulate in vital tissues and organs, suggesting a significant health risk (Han et al., 2024), as they have been classified as endocrine disruptors (Wu et al., 2023). Despite the growing interest in PS-NP toxicity, knowledge of their specific impact on the female reproductive system, particularly during gestation, remains limited (Adhikari et al., 2024). Our study investigated the effect of oral gestational exposure to PS-NPs (100 nm; 1000 µg/day for 22 days) on uterine histomorphology in F0 Wistar rats. We recorded the number of pups born, maternal body weight, and offspring body weight at birth and at postnatal day 12. Histomorphological analysis of the uterus was performed using Masson's trichrome staining to assess endometrial epithelial and muscular layer thickness, glandular diameter, and vascular density. Results. Gestational exposure to PS-NPs did not affect fertility (number of pups born), maternal body weight, or offspring birth weight. However, on postnatal day 12, the weight of offspring prenatally exposed to PS-NPs was higher. Histologically, PS-NP-exposed F0 rats exhibited uteri with larger diameters, reduced muscular and epithelial layer thickness, and lower densities of uterine glands and blood vessels. These findings suggest that PS-NPs impair cellular proliferation and vascularization processes, disrupting the uterine microenvironment required for adequate gestation, which could ultimately compromise reproductive physiology.

SP 29. *Evaluation of the Effect of Nopal Extract on a Spheroid Model of Colon Cancer Cells*

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Colon cancer ranks third among the most lethal neoplasms in the world, and in Mexico, it is the second most lethal type of cancer. One of the risk factors is the high consumption of red and/or processed meats; therefore, a healthy diet can reduce this risk. The prickly pear cactus (Opuntia ficus-indica) is an edible cactus, and various studies have shown the biological potential of its components against different types of cancer. Previous studies by the task force suggest that polyphenol-rich cactus extracts can induce cell death in two-dimensional cultures of colon cancer cells. However, spheroid cultures of cancer cells more accurately mimic tumor growth, considering the cell organization in a three-dimensional way, and allow the efficacy of drugs and treatments to be studied more accurately. The objective of this research was to generate a spheroid model of colon cancer cells and to evaluate the effect of prickly pear extract on cell viability, considering the IC50 reported for two-dimensional cultures. To this end, cell viability experiments were conducted in two-dimensional cultures of Caco-2 cells at varying concentrations to determine the IC50. Additionally, the number of Caco-2 cells required to form a spheroid was assessed. Preliminary results indicate that the IC50 is 2mg/mL in two-dimensional culture, which is consistent with previous studies. Additionally, the initial results suggest that the number of seeded cells enables the formation of spheroids with a diameter of approximately 100 µm. However, the results suggest that a larger number of cells needs to be seeded. These results lay the foundations for studying the effect of prickly pear extract on a spheroid model of colon cancer cells.

SP 30. *Multi-Omics Analysis of a New Rat Metastatic Hepatocellular Carcinoma Model*

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Approximately 70% of patients with hepatocellular carcinoma (HCC) die from advanced stages of the disease (metastasis) mainly because of current therapy failure. One of the main causes of treatment ineffectiveness is the lack of in vivo models closely representing the biology of patients with metastatic HCC, since they lack homotypic interactions and a complete immune system. To address these, we developed two syngeneic transplantation HCC models, a non-metastatic (Samy) and a metastatic (Samy Met). The aim of this study is to perform their multi-omic characterization, to understand the biology of this disease, as well as to identify prognostic biomarkers and new therapeutic targets. Tran-

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scriptome and proteome were obtained from primary tumors of both models and analyses clearly separated the two models, demonstrating their distinct behavior. The overexpressed genes in the metastatic model are associated with poor prognosis, metastasis, and immune system evasion, while the underexpressed genes are related to good prognosis and tumor-suppressive capacities. This same pattern was confirmed in proteins with differential abundance, validating our models. To select genes with a potential prognostic capacity, genes within GSEA enriched gene sets were compared with the overabundant proteins, and 96 genes overlapped. Within these, the expression of four upregulated (FGA, KNG1, GLDC and CDH1) and two downregulated (DNM1 and MMP9) genes is associated to higher metastasis risk and poor prognosis in patients with HCC, using the TCGA-LIHC and GSE14520 cohorts. We propose them as a metastasis signature, however in vitro and in vivo experiments are needed to determine whether these are potential therapeutic targets in metastatic HCC.

SP 31. *Small Particles, Big Epigenetic Effects: PM2.5 Pollutants in Breast Cancer Cells*

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Air pollution represents a significant challenge to public health. Among the various pollutants, urban particulate matter (UPM) is considered particularly harmful, as it is a complex mixture of organic and inorganic compounds whose toxicity depends on size and composition. The finer particles, such as PM2.5, pose a greater risk since they can reach the pulmonary alveoli and enter the bloodstream. These particles contain an organic fraction composed of hydrocarbons and carbon, and an inorganic fraction that includes heavy metals, conferring genotoxic and mutagenic properties. Exposure to PM2.5 is associated with chronic diseases, carcinogenic processes, and neurological alterations. Although most studies focus on pulmonary cell lines, little is known about its effects on mammary cells, such as the human breast cancer cell line MCF-7, a triple-negative breast cancer model, and no data exist at the continental level. This study aimed to evaluate the cytotoxic and phenotypic effects on MCF-7 cells exposed to PM2.5, as well as to correlate epigenetic alterations induced in tissues associated with breast cancer metastasis through transcriptomic analysis in genomic reservoirs. Air samples were collected using high-efficiency filters placed at monitoring stations in Puebla by the Secretaría de Medio Ambiente del Estado de Puebla. The two fractions of PM2.5 were purified using ultrasonication, and MCF-7 cells were then acutely exposed to the inorganic fraction at different doses for 24 h. We implemented the MTT assay to evaluate cell viability, obtaining a dose–response curve to calculate IC50 and IC10 values, in addition to recording morphological changes through bright-field imaging assays. Our findings show that the organic and inorganic fractions of PM2.5 exert differential effects on the minimum dose responsible for inducing morphological and viability alterations in human MCF-7 cells. For the inorganic fraction, a significant decrease in cellular functionality was observed, verified by both viability assays and aberrant morphologies, with enlarged cell bodies and poor adhesion. Understanding the morphological and cytotoxic changes derived from acute and chronic UPM exposure, both at regional and local levels, may provide crucial evidence for risk assessment and for strengthening public health prevention strategies.

SP 32. *Sensitization of Sorafenib-Resistant Hepatocellular Carcinoma Cells by Bovine Lactoferrin: An Effect on the Regulation of Long Non-Coding and Messenger RNAs (lncRNA and mRNA)*

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Hepatocellular carcinoma (HCC) is among the most lethal cancers worldwide, with high mortality largely due to late-stage diagnosis and limited therapeutic options. Sorafenib, a first-line treatment for advanced HCC, often loses efficacy as resistance develops, severely restricting its long-term benefits. This challenge has prompted efforts to identify strategies capable of overcoming drug resistance and improving patient outcomes. In this study, we investigated bovine lactoferrin (bLf) as a potential sensitizer of sorafenib-resistant HCC cells. Lactoferrin (Lf) is a glycoprotein of the innate immune system; bovine Lf (bLf), functionally comparable to human Lf, is safe for human use and displays anticancer properties, including synergy with chemotherapy. We established a resistant Hep3B cell line (Hep3B-R) through repeated drug exposure and confirmed increased tolerance by IC50 analysis. Treatment of Hep3B-R cells

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with sorafenib plus iron-saturated bLf (holo-bLf) restored sensitivity to levels comparable to parental Hep3B cells. To explore underlying mechanisms, RNA sequencing revealed marked transcriptional differences between resistant and non-resistant cells, as well as reversal of resistance-associated patterns after holo-bLf treatment. Gene Set Enrichment Analysis (GSEA) identified ferroptosis as a pathway enriched in holo-bLf–treated resistant cells. In vivo confocal microscopy confirmed induction of ferroptosis by holo-bLf. Further analyses highlighted the ATF3/HMOX1/GPX4 axis, a pathway involved in ferroptosis and oxidative stress, as a potential mediator of holo-bLf effects. Among differentially expressed lncRNAs, LINC02015 emerged as a candidate regulator of ferroptosis, correlating with ferroptosis-related genes such as HMOX1, which is central to oxidative stress and iron metabolism. In conclusion, our findings suggest that holo-bLf restores sorafenib sensitivity in resistant HCC cells by inducing ferroptosis, implicating the LINC02015/ATF3/HMOX1/GPX4 axis as a therapeutic target. This approach offers a promising strategy for enhancing treatment outcomes in advanced HCC.

SP33. *Determination of Gram (-) Bacteria Associated With Atmospheric Particles (PM10) Indoors in the City of* San Francisco de Campeche

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SP 34. *Exposure to Atoyac River Water Reduces Longevity and Reproductive Capacity in Caenorhabditis elegans*

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The Atoyac River is one of the most polluted water bodies in Mexico, receiving untreated discharges. Chronic exposure to its contaminants poses risks to both ecosystems and human health. Within the framework of the human exposome, which integrates all exposures throughout the lifespan, the use of biological models such as Caenorhabditis elegans enables evaluations in shorter time frames, representing an advantage over other experimental organisms. The Upper Atoyac Basin has been classified as a region of socio-environmental emergency; however, its impact on present and future generations remains incompletely understood. Identifying long-term effects in human populations would require cohort studies spanning decades. In this regard, the use of animal models accelerates the generation of evidence. The nematode C. elegans, an in vivo model in toxicology, is particularly suitable for evaluating reproduction and longevity (Che-Trejo et al., 2025). To assess the effects of exposure to Atoyac River water on reproductive and longevity parameters in C elegans. Water samples were collected from the Atoyac River (19.0709680 N, 98.2399340 W, Puebla, Mexico). The water was filtered; the experimental group (RA) was cultured in media prepared with Atoyac water, while the control group (CO) was cultured with distilled water. Parameters such as egg laying, egg-laying rate, and in utero embryos were analyzed by microscopy. Longevity was assessed by daily counting of dead animals over four weeks using Kaplan-Meier survival curves. RA nematodes exhibited reduced oviposition, egg-laying rate, and egg retention compared to the CO group. Longevity was also decreased (RA: ±15 days, CO: days), suggesting potential consequences for health and life expectancy in exposed populations.

SP 35. *Bifidobacteria Attenuate High Fat Diet-Induced Changes in the Rat Liver and Cecum*

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Liver and cecum have bidirectional communication that is crucial for metabolic homeostasis and its disruption is associated with metabolic dysfunction-associated steatotic liver disease (MASLD). Diets high in saturated fats disrupt the cecal environment, altering intestinal permeability and increasing endotoxin levels, which contribute to MASLD

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progression. Here, we determined whether bifidobacteria supplementation may therefore serve as an effective strategy to protect the liver and maintain gut – liver homeostasis in a rat model subject to high-fat diet (HFD). To investigate the molecular and histological effects of bifidobacteria spp on HFD-induced hepatic and intestinal damage, an animal model was established. Wistar rats were fed a high-fat diet for ten weeks. Animals were intragastric administrated with 1X109 UFC of bifidobacteria spp, every other day. Liver and cecum tissues were collected for histological analyses, immunofluorescence, and Western blot techniques. HFD administration increased fat accumulation in the liver, as well as protein adducts formation, as detected by 4HNE-protein binding level. Supplementation with bifidobacteria spp. reduced hepatic fat deposition accompanied by modifications in the 4HNE-protein binding pattern. In cecum, the bifidobacteria spp administration restored both OCLN and TLR4 protein levels.
Bifidobacteria spp. administration induced significant changes in both liver and cecum. In the liver, it reduced fat accumulation, suggesting a metabolic protective effect; also, altering 4HNE-protein binding pattern. In the cecum, the restoration of OCLN and TLR4 protein levels strongly suggests that bifidobacteria reinforce gut barrier integrity and modulate immune responses. These findings indicate that Bifidobacteria spp is effective in mitigating HFD-induced hepatic steatosis and gut dysfunction, potentially, via microbiota–gut–liver axis regulation.

SP 36. Asociación entre maduración somática, estado nutricional y pico de velocidad de crecimiento en adolescentes de Campeche.

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SP 37. *Pan-Tissue Effects of PM2.5 Pollutants by Transcriptomics and Epigenomics Analysis*

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Increasing air pollution, especially fine particulate matter (PM2.5), raises serious public health concerns due to its association with a variety of diseases. Our study explores the effects of PM2.5 particles on a variety of human cell lines, aiming to better understand the underlying molecular mechanisms of adverse health effects relying on global multiOMIC profiles by RNA-Seq and Methyl-Seq (RRBS) of human cell lines exposed to PM2.5. Due to concerning environmental reports for PM2.5 air concentrations in recent years, we also collected and analyzed PM2.5 emission data from 2015 to 2022 in 5 different monitoring stations to visualize significant environmental trends in Puebla City, Mexico. RNA-sequencing and Methyl-Seq (RRBS) data from human cell lines of different tissues (trophoblast cell line HTR8/SVneo, GSE237795; kidney proximal tubular epithelial cell line HK-2, GSE208331; lung epithelial cell line BEAS-2B, GSE182199), which were exposed to different concentrations of PM2.5, were analyzed to identify genes and pathways affected by PM2.5 exposure using nextflow bioinformatics pipelines. Our preliminary analyses reveal that PM2.5 influences conserved biological pathways related to oxidative stress and mitochondrial apoptosis, autophagy and necroptosis, glycolysis, and cell migration. Additionally, the visualization of PM2.5 emission data for Puebla from 2015 to 2022 shows a trend of annual values consistently exceeding the limits set by the Mexican Official Standard NOM-025-SSA1-2021, and those suggested by the World Health Organizations for PM2.5 and PM10 in the atmosphere at 10 and 20 µg/m3, respectively. This study will provide crucial insights into the molecular effects of PM2.5 particles on human health, as well as insights into PM2.5 emission trends over time. These findings could have important implications for the development of pollution control policies and public health strategies aimed at mitigating the adverse effects of air pollution on the population.

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SP 38. *Beyond the Infection: The Role of HRV in Detecting Post-COVID-19 Cardiac Autonomic Dysfunction*

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COVID-19 infection could cause "Long COVID syndrome". An important sequela of this is the cardiac dysautonomia which is an Autonomous Nervous System (ANS) affection evidenced by a decrease in the heart rate variability (HRV) that can be evaluated by the pNN50 indicator, values equal to zero indicate absence of normal heart beat fluctuations. There's very limited evidence of diagnosis, follow up and management of young adults with a COVID -19 induced cardiac dysautonomia through standardized protocols. To describe a series of clinical cases of patients with sequelae because of SARS-CoV-2 reflected in a HRV decrease as an ANS affection marker. Through an observational study of HRV in a 100 medicine students population (18-24 years old), excluding volunteers with cardiac pathology or use of toxic substances. Our sample was classified in 46 controls (no COVID-19) and 54 SARS-CoV-2 positives. Three SARS-COV-2 positive cases were isolated with a pNN50 indicator of 0, these patients received a follow up with no pharmacological recommendations (cardiovascular exercise, hidratación, and BMI control). In all three cases, low values for RMSSD, NN50, PNN50, and SD1 were observed, which reflects a decrease in parasympathetic tone, leading to low heart rate variability. An increase in these parameters was observed at the nine-month follow-up. However, symptoms such as fatigue and orthostatic dizziness persisted in the two patients who were overweight and sedentary, while the patient with regular physical activity showed an absence of clinical manifestations. Long COVID induced cardiac dysautonomia and its severity is confirmed in this study. Opportune treatment and the attention to this condition are fundamental in the future complications prevention. Cardiac dysautonomia's no pharmacological treatment results in a significant improvement of the analyzed HRV indicators.

SP 39. *Epigenomic Analysis Induced by Ionizing Radiation in an Ex Vivo 3D Model (PTCS) of Murine Brains*

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We know that up to 70% of cancer patients will receive radiation therapy as a treatment, so it is surprising that it is still so unspecific. The effectiveness of radiotherapy is influenced by the tumor microenvironment, so preclinical research faces challenges in replicating these environments in vivo and thus identifying the precise molecular mechanisms, since the 2D cell culture does not manage to recapitulate the physiological context of the microenvironment. Currently, we have opted for the implementation of precisely cut tissue slices (PTCs), which are three-dimensional tissue explants derived from organs that can be grown ex vivo. To analyze markers of epigenomic alterations induced by ionizing radiation in 3D tissues of murine brains. Three 4-week-old male Wistar rats were maintained with a light/dark cycle of 12 hours, at 22+/-2°C, and fed ad libitum. The brains were cut at 400 µm using a VF-310-0Z vibratom (Compresstome), these were distributed in plates of 48 wells and irradiated with 2.66 Gy or 3 Gy. The PTCs were punched and incubated again at 37 °C with 5 % CO2, for cell viability analysis over 5 consecutive days. In addition, differential expression analysis with radio-responsive or radio-resistant genes was performed to assess the associated molecular mechanism. We validated the differential expression of a set of radio-sensitive genes (MUS81, DNAJC17) and radioresistant genes (SP1, VAV2), as well as tissue viability under implementation of optimized PCTs protocols. The ex vivo models by means of PCTs in murine models are optimal to characterize the epigenetic mechanisms, potentially reprogramming, associated with radio-responsiveness or radio-resistance, a phenomenon of high clinical importance in cancer patients.

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SP 40. *Protein-Protein Interactions Between pRb and the RXRα/β and TRα/β Receptors During Limb Regeneration in Ambystoma mexicanum*

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Ambystoma mexicanum possesses an exceptional regenerative capacity to regenerate limbs through epimorphosis, a process that depends on precise cellular reprogramming, regulating the dynamics between proliferation and differentiation states. In this regard, the retinoblastoma protein (pRb) plays an essential role in regulating the cell cycle. Furthermore, pRb interacts with various proteins through canonical and non-canonical (LxCxE/LxCxD) motifs. RXRΔΔ and TRΔΔreceptors have been reported to display these motifs, making them potential pRb interactors. The objective of this study was to determine whether pRb interacts with RXRα/β and TRα/β through these motifs and whether they co-localize in specific tissues and stages during limb regeneration. To evaluate this interaction in vivo, the Bimolecular Fluorescence Complementation (BiFC) system was used. To confirm the relevance of the motifs, AxAxA mutants were generated by site-directed mutagenesis. Furthermore, tissue from different stages of limb regeneration was used, gene expression levels of AmTRa and AmpRb were analyzed by qRT-PCR, and colocalization dynamics were assessed by immunofluorescence and confocal microscopy. The results show predominantly nuclear and, to a lesser extent, cytosolic interaction in wild-type proteins. The mutation in the binding motif significantly reduced the fluorescence intensity, indicating an important role of the motif in the interaction. We observed an increase in the expression levels of AmTRa and AmpRb transcripts from the late blastema stage to redifferentiation, consistent with the nuclear colocalization observed in regenerative tissue. Together, these results suggest that the interaction between AmTRa and AmpRb could play a role during key stages of limb regeneration.

SP 41. *The Impact of Arpin on Prognosis and Invasion in B-Cell Acute Lymphoblastic Leukemia*

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Arpin is a recently discovered protein that functions as a negative regulator and inhibitor of the Arp2/3 complex. This complex is a critical driver for the nucleation of new actin filaments, the formation of lamellipodia, and the initiation of cell migration.

Problem Statement. B-cell Acute Lymphoblastic Leukemia (B-ALL) is the most prevalent cancer in children. It is a heterogeneous disease characterized by a significant burden of chromosomal mutations and genetic alterations. Evidence suggests that the clonal proliferation in B-ALL is driven by the dysregulation of the bone marrow niche and its components, including an increased production of pro-inflammatory cytokines. To determine the relationship between Arpin expression levels and disease progression in patients with B-cell Acute Lymphoblastic Leukemia. To identify whether Arpin can be considered a novel prognostic biomarker and a potential therapeutic target for B-cell Acute Lymphoblastic Leukemia. Bone marrow samples were collected from adult patients (>18 years) with Acute Lymphoblastic Leukemia and from healthy donors at the National Medical Center (CMN) "La Raza" of the Mexican Social Security Institute (IMSS). *The study involved a series of experiments, including Western Blot analysis, lymphocyte cell migration assays, and spectral flow cytometry. The results revealed a significant decrease in Arpin expression in patients with Acute Lymphoblastic Leukemia compared to healthy controls. In B-ALL, migratory capacity is directly linked to prognosis and plays a pivotal role in disease relapse. Therefore, the observed reduction of Arpin in B-ALL could be associated with patient prognosis. These findings suggest that Arpin may represent a novel biomarker and a promising therapeutic target for future clinical applications.*

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SP 42. *Evaluation of Epigenetic Mechanisms in Breast Cancer Cells That Survive Photodynamic Therapy*

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Triple-negative breast cancer (TNBC) is clinically aggressive and has limited therapeutic options due to the absence of receptors such as ER/PR/HER2. Photodynamic therapy (PDT) has been used as an alternative for disease control by stimulating a photosensitizer and generating reactive oxygen species. However, studies on the epigenetic regulatory mechanisms that maintain cell population survival after PDT application have not yet been conducted. To address this issue, stress-responsive genes and epigenetic changes that facilitate post-PDT survival were evaluated in a TNBC model. Cell models subjected to PDT were combined with transcriptomic profiles of chromatin expression and accessibility, complemented by validations targeting the potential mechanisms involved. Overall, preliminary results indicate the coordinated activation of antioxidant, inflammatory, and hypoxic responses, along with chromatin remodeling involving histone demethylases. Based on these findings, the validation of epigenetic mechanisms that promote survival to targeted therapies is planned, in order to support possible therapeutic combinations aimed at reducing this cellular capacity.

SP 43. *What COVID Left Us: Sympathetic-Vagal Imbalance in Young University Students*

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COVID-19 is a disease that affects worldwide and causes mainly pulmonary manifestations. If these symptoms persist in the post-acute phase, it is called long COVID syndrome, its ANS affection can cause cardiac dysautonomia. There is very few evidence about long COVID´s impact on young adults without comorbidities, since most of the studies evaluating its effect on ANS through the heart rate variability analysis are focused on elderly and/or adults with comorbidities. To evaluate long COVID´s effects on heart autonomic regulation on college students. We did an observational study on 79 medicine students (18-24 years old) from BUAP: 35 had no previous SARS- CoV-2 infection (control group) and 44 with long COVID (COVID group). Patients with heart disease, diabetes, use of toxic substances or BMI >25. The HRV was analyzed through an electrocardiogram on time, frequency and geometric domains. In the time domain analysis, the COVID group had lower values compared to the control group in DERR, RMSSD, NN50 and PNN50 metrics. In the frequency domain we observed higher values in the control group in high frequency waves, while in low frequency waves, and in the high frequency to low frequency waves ratio predominates the COVID group. Finally, the geometric index showed a decrease of the long and short term HRV in the COVID group. Heart dysautonomia is a long COVID sequel in young adults without previous heart diseases. Long COVID produces an increase of the resting heart rate.

SP 44. *Role of SNORD44 in Modulating the Expression of Caspases-8, -9 and -3 in the REH Cell Line*

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Acute lymphoblastic leukemia (ALL) is the most common cancer in childhood. The survival rate of patients with ALL in developed countries reaches up to 80%, however, in countries such as Mexico, it is usually only 40%. Despite advances in treatments, the relapse rate in pediatric patients with ALL remains between 15% and 20%. The prevalence, low survival, and relapse rate show the imperative need to study ALL molecularly. Previous studies in our research group have demonstrated the dysregulation of some small nucleolar RNAs (snoRNAs, genes involved in ribosomal biogenesis) in patients with ALL, including SNORD44. In preliminary studies, it has also been identified that SNORD44 overexpression increases proliferation and decreases apoptosis in the REH leukemia cell line; then, the objective of this work was to evaluate the role of SNORD44 on the regulation of caspase -8, -9, and -3 expression. To do so, the REH cell line was transfected with either a SNORD44 expression plasmid (four independent experiments) or with siRNA directed against

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SNORD44 (three independent experiments). After verifying the efficiency of silencing or overexpression, the relative expression of caspases -8, -9, and -3 was quantified by qRT-PCR. The results revealed that SNORD44 silencing significantly decreased caspase-9 expression; on the other hand, the overexpression of SNORD44 resulted in a tendency to increase caspase-9, although these results were not significant. In conclusion, preliminary results suggest that SNORD44 could play a regulatory role in apoptosis, specifically through the regulation of caspase 9.

SP 45. *Effect of synthetic methylxanthines and caffeine supplementation on premature infant mortality and apnea control.*

Grecia Guadalupe Rodríguez Paredes, Sonia Muñoz Cerón.

Premature apnea is one of the most frequent complications in neonates with a gestational age <37 weeks, primarily caused by immaturity of the central nervous and respiratory systems. It is characterized by respiratory pauses ≥20 seconds, associated with bradycardia, hypoxemia, and cyanosis. Its pathophysiology involves poor neuronal myelination, ineffective biphasic ventilatory response to hypoxia and hypercapnia, and weakness of thoracic and diaphragmatic muscles, which promotes alveolar collapse and recurrent desaturation episodes. These alterations directly impact neonatal morbidity and mortality, potentially leading to neurological sequelae, bronchopulmonary dysplasia, and prolonged hospital stays. An observational, cross-sectional, and retrospective study was conducted in the NICU of the General Hospital of Tlaxcala through the review of medical records of preterm infants with apnea from January 2019 to November 2021. The sample was divided into three groups: untreated, treated with caffeine supplementation, and treated with synthetic methylxanthines. Clinical variables evaluated included survival, need for mechanical ventilation, associated complications, and length of hospitalization. Caffeine, as a non-selective adenosine receptor antagonist, stimulates the respiratory center, increases minute ventilation, improves diaphragmatic contractility, and reduces hypoxic depression, with a better safety profile compared to other methylxanthines. Its use is expected to significantly decrease the frequency of apnea episodes, reduce mortality, and improve the clinical course of neonates. Conclusions: Caffeine emerges as the first-choice treatment for premature apnea, combining efficacy, safety, and accessibility. Its implementation in hospital protocols can optimize the management of respiratory immaturity, reduce complications, and help improve neonatal health indicators in resource-limited settings.

SP 55. *Genomics in Gynecologic Cancer in Mexico: Towards the Era of Precision Oncology*

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Gynecologic cancers (ovary, endometrium, and cervix) represent a significant cause of morbidity and mortality in Mexico. Next-generation sequencing (NGS) has become a fundamental tool to identify genomic alterations with potential diagnostic, prognostic, and therapeutic impact. However, in the Mexican population, there is still a gap in systematic molecular characterization. This study aimed to determine the frequency and profile of somatic and germline mutations detected by NGS in patients with gynecologic cancers treated at a private oncology center in Puebla. An observational, descriptive, cross-sectional study was conducted including 48 patients with histologically confirmed gynecologic cancer. Clinical, histopathological, and molecular variables were analyzed using somatic NGS panels (Foundation Medicine, Caris, Axiona, Guardant 360) and germline panels (Invitae). Statistical analysis was performed with Jamovi 2.6.25. Of the patients, 52.1% had ovarian cancer (n=25), 27.1% cervical cancer (n=13), and 20.8% endometrial cancer (n=10). A family history of cancer was reported in 71.1% of cases. Most patients (92.1%) were diagnosed at advanced stages (III–IV). The most frequent somatic mutations were TP53 (43.9%), PIK3CA (17.1%), PTEN (14.6%), BRCA1 (9.8%), and ERBB2 (9.8%). At the germline level, BRCA1 was the most common (15.4%). Overall, 58.5% of patients harbored clinically actionable mutations, making them eligible for targeted therapies (e.g., PARP inhibitors, immunotherapy). This study represents one of the first systematic analyses in the Mexican population, confirming the high prevalence of actionable mutations in gynecologic cancers. The findings reinforce the need to integrate genomics into clinical practice to advance precision oncology in Mexico.

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SP 56. *Assessment of the Antifungal Activity of Green-Synthesized Silver Nanoparticles Against Clinical Strains of Candida albicans*

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Infections caused by Candida albicans represent a significant clinical challenge due to the growing number of strains resistant to commercial antifungal agents. In this context, silver nanoparticles (AgNPs) synthesized using aqueous plant extracts have emerged as a promising strategy, combining sustainability principles with notable antifungal potential. The minimum inhibitory concentration (MIC) is a standard parameter in antifungal susceptibility assays; however, it does not always accurately reflect the true efficacy of treatments. Therefore, complementary metrics are needed to assess both fungistatic and fungicidal activities. To evaluate the antifungal activity of AgNPs synthesized with aqueous extracts of Pyracantha koidzumii (AgNPs-P) and Schinus molle (AgNPs-S) against clinical isolates of Candida albicans, in comparison with a reference ATCC strain, using parameters beyond MIC. AgNPs were synthesized using aqueous leaf extracts and characterized by UV–Vis spectroscopy, FTIR, DLS, NTA, and TEM. Susceptibility assays were conducted to determine the MIC, minimum fungistatic concentration (MFuC), and minimum fungicidal concentration (MFC). Additionally, the particle efficiency index (PEI) was calculated, defined as the number of nanoparticles required to eliminate one colony-forming unit (CFU). Both types of AgNPs exhibited a MIC of 1.56 µg/mL; however, AgNPs-P demonstrated superior antifungal activity, with lower MFuC (6.25 µg/mL) and MFC (12.5 µg/mL) values compared to AgNPs-S, as well a reduced PEI of 2.8 × 104 NPs/CFU. Overall, AgNPs can induce cell wall damage, inhibit hyphal development, and reduce proteolytic activity, positioning AgNPs-P as a promising therapeutic candidate.

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CR 1. *Tuberculous Meningitis: A Diagnostic Challenge. Case Report*

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Tuberculous meningitis is the most severe form of extrapulmonary tuberculosis, constituting a medical emergency with high morbidity and mortality, especially in endemic countries and in immunocompromised patients. It results from the invasion of the central nervous system by Mycobacterium tuberculosis, causing inflammation of the basal meninges, vascular compromise, and alterations in the cerebrospinal fluid. This is a clinical case of a 19-year-old male patient from Jalpa, Tabasco, a student weighing 31 kg, with no relevant medical or epidemiological history. He was HIV-negative. In February 2025, he began experiencing intermittent holocranial headaches that subsided with paracetamol. On March 22, he presented with severe headache and fever; he received unspecified treatment without improvement. He was treated at the General Hospital of Jalpa de Méndez and discharged with outpatient treatment. Four days later, he returned with a 10/10 headache, fever, and a tonic-clonic seizure, and was referred to General Hospital Zone 46 in Villahermosa due to suspected subarachnoid hemorrhage, which was later ruled out. He was transferred to General Hospital Zone 2-A on March 26. During his stay, he showed neurological deterioration, with positive Brudzinski and Babinski signs. An MRI on April 3 suggested an infectious process compatible with tuberculosis meningitis versus cysticercosis, and albendazole and dexamethasone were initiated. On April 7, Epidemiology ordered cerebrospinal fluid analysis with GeneXpert. On April 15, tuberculosis meningitis with rifampicin resistance was confirmed, and antituberculosis treatment was initiated under the TAES regimen. Tuberculosis meningitis, although less frequent than pulmonary tuberculosis, is one of the most lethal presentations. It primarily affects individuals in endemic countries and vulnerable patients, with high mortality and neurological sequelae in survivors. Early diagnosis and timely treatment are crucial for reducing complications and improving survival. Although early diagnosis and appropriate treatment can significantly improve survival rates and reduce sequelae, delays in detection and treatment remain a challenge.

CR 2. *Management of Subtrochanteric Fracture with Pseudoarthrosis in an Osteogenesis Imperfecta Patient Using 3D Planning and PHILOS Plate Placement in a Trauma and Orthopedics Hospital*

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Osteogenesis imperfecta (OI) is a hereditary bone dysplasia caused by a collagen type I synthesis disorder, leading to severe bone fragility, progressive deformity, and a high risk of fractures. The global incidence is approximately 1 in 10,000 live births, while in Mexico, the reported incidence is between 1 in 10,000 to 25,000 births. In adults with OI, fractures of long bones, particularly in the diaphysis, show a higher prevalence of pseudoarthrosis, which makes their surgical management a significant challenge. We present the case of a 31-year-old female patient with a prior diagnosis of osteogenesis imperfecta, who came to our unit with a pseudoarthrosis in the middle third of her right femur and a subtrochanteric fracture. To optimize planning and address the case's complexity, we used 3D modeling and printing of the affected limb as a support tool. This model allowed us to precisely simulate the osteotomy for angular correction and implant placement. A successful external closing wedge osteotomy was performed, followed by fixation with a PHILOS anatomical plate, which was chosen based on the patient's bone characteristics and the availability of surgical materials in our unit. The coexistence of a subtrochanteric fracture and femoral pseudoarthrosis in an adult patient with osteogenesis imperfecta is a complex and uncommon clinical scenario. This case demonstrates that preoperative surgical planning with 3D technology is a valuable tool for managing such complex injuries. Our experience suggests that conservative treatment of femoral fractures in adults with OI carries a high risk of non-union, making a meticulous surgical strategy key to successful treatment.

CR 3. *Divergent Outcomes in Two Patients With Intestinal-Type Ampullary Carcinoma: The Role of Genomic Profiling*

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Ampullary adenocarcinoma is a rare gastrointestinal neoplasm characterized by remarkable molecular heterogeneity, which complicates its therapeutic management. Although treatment is often guided by clinical and histological criteria,

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recent research has highlighted the relevance of genomic profiling for improved prognostic stratification and treatment selection. We report two cases of intestinal-type ampullary adenocarcinoma, both stage IIIB, harboring a KRAS G12D mutation. The first case exhibited additional TP53 mutations and SMAD4 loss, with an overall survival of 39 months, while the second presented alterations in APC and BRAF F595L, with an overall survival of 51 months. Both patients received fluoropyrimidine-based adjuvant chemotherapy. Despite sharing the same stage and histological subtype, they experienced divergent clinical outcomes, likely attributable to differences in their molecular profiles. This report underscores the value of genomic profiling in the management of ampullary cancer, particularly in underrepresented populations such as the Mexican cohort.

CR 4. *Prader-Willi Syndrome Due to Altered Methylation Pattern: A Case Report*

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Prader-Willi syndrome (PWS) is a multisystemic neurobehavioral disorder classified as a rare disease, with an approximate incidence of 1 in 10,000 to 1 in 30,000 births. Clinically, it manifests in newborns with severe hypotonia and feeding difficulties. In childhood, hyperphagia leads to morbid obesity, accompanied by respiratory and cardiac complications. Patients present with behavioral disorders, mild-to-moderate intellectual disability, and an increased risk of psychosis in adulthood. At the molecular level, PWS is caused by alterations in the imprinted region 15q11.2-13.3, due to paternal deletion (65-70%), maternal uniparental disomy (20-25%), or imprinting defects (1-3%). PWS remains underdiagnosed due to the variability in clinical presentation and a lack of awareness of the disease among healthcare professionals. This case aims to illustrate the value of timely clinical and molecular diagnosis. We describe a 5-year-old female patient, the product of a second pregnancy, weighing 2,820 g and measuring 51 cm at birth. From the first weeks of life, she presented with generalized hypotonia, poor sucking, and weight loss. At 56 days, she exhibited microcephaly, facial dysmorphisms, and psychomotor delay. At 2 years of age, she developed hyperphagia, obesity, and obstructive sleep apnea. At 4 years of age, methylation-sensitive PCR was performed, revealing an altered methylation pattern of the SRNPN gene, confirming Prader-Willi syndrome (PWS). A microarray study ruled out deletion of the implicated region. These findings highlight the importance of the differential expression of imprinted genes, in this case, the lack of expression of the paternal allele genes with double expression of the maternal allele genes, controlled by epigenetic mechanisms such as methylation. Early knowledge and diagnosis of this condition through appropriate molecular studies allows for the implementation of multidisciplinary treatment that improves the quality of life and prognosis of patients.

CR 5. *Agenesis of the Corpus Callosum, Ocular, Genital and Cardiac Syndrome: Case Report*

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Agenesis of the corpus callosum, ocular, genital, and cardiac syndrome (ACC-OGC) is a rare genetic condition with significant phenotypic variability associated with a mutation in the CDH2 gene on chromosome 18q12. This gene encodes the N-cadherin protein, which is essential for neuronal migration and tissue formation during embryonic development. The mutation results in a defective protein, leading to the characteristic malformations of this syndrome. Information on ACC-OGC syndrome is very limited and based on isolated case reports, making it a poorly understood medical entity. The exact causes, long-term prognosis, and specific management protocols have not yet been defined. The scarcity of studies prevents the establishment of robust, evidence-based management guidelines. We report a familial case of two sisters; the younger sister presented with a clinical picture consistent with the symptoms reported for ACC-OGC syndrome, including agenesis of the corpus callosum, developmental delay, intellectual disability, and various craniofacial anomalies. Likewise, the older sister also met some criteria for the syndrome, but presented with vertebral anomalies not previously reported in this condition. Genetic analysis revealed that both sisters carry a variant of uncertain significance in the CDH2 gene, c.36_56DUP (P.PRO13_LEU19DUP). A segregation study of the parents confirmed that the father is a carrier of the variant, establishing an autosomal dominant inheritance pattern with incomplete penetrance. ACC-OGC syndrome is a cause of one of the most common brain malformations. Its diagnosis is essential for detecting associated malformations, allowing for early therapeutic intervention and improving the prognosis. Future research is indispensable to close the knowledge gap and establish standardized and effective clinical management.

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CR 6. *Case Report: Tetralogy of Fallot Due to 22q11.2 Microdeletion*

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Tetralogy of Fallot (TOF) is a prevalent cyanotic congenital heart defect, accounting for approximately 3.5% of all cardiac malformations, with an estimated incidence of 0.28 per 1,000 live births. The tetrad of anomalies comprising its pathophysiology are a ventricular septal defect, obstructive pulmonary stenosis, overriding aorta, and secondary right ventricular hypertrophy. Its etiology is divided into syndromic and non-syndromic forms. The syndromic form is primarily associated with the 22q11.2 microdeletion, which arises from non-allelic homologous recombination. This deletion affects a crucial genomic segment that includes the TBX1 gene. The non-syndromic form is linked to pathogenic variants in key cardiogenesis genes, notably NOTCH1, FLT4, and the transcription factor GATA4. The clinical case of a 10-month-old male with a diagnosis of TOF confirmed by echocardiogram is analyzed. Physical examination revealed a broad forehead, almond-shaped eyes, horizontal palpebral fissures, a broad nasal pyramid and root with a bulbous tip, an intact oral cavity, micrognathia, borderline ear placement, a grade IV chest murmur, a soft, depressible abdomen, male genitalia with a testicle in the scrotal sac, and upper extremities with a left hand exhibiting a transverse palmar crease. Genetic analysis was performed using the FISH technique with the TUPLE1 probe, yielding the result: 46,XY[30]. ish del22(q11.2q11.2)(TUPLE1-,SHANK3+)[10].nuc ish(TUPLE1x1)[100], confirming the syndromic etiology and the 22q11.2 microdeletion. Genetic alterations are one of the main determinants in the development of congenital heart disease. In particular, the 22q11.2 microdeletion has been associated with various cardiac malformations, including Tetralogy of Fallot, as evidenced in this case. This finding underscores the importance of incorporating genetic analysis into the clinical evaluation of patients with suspected or diagnosed congenital heart disease, in order to promptly identify chromosomal abnormalities that contribute to a comprehensive diagnostic, prognostic, and therapeutic approach.

CR 7. *Intellectual Developmental Disorder, Autosomal Dominant Type 38, Caused by a New Variant in EEF1A2: Case Report and Literature Review*

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Mutations in the EEF1A2 gene are associated with a rare neurodevelopmental disorder characterized mainly by the presence of dysmorphic facial features, early-onset epilepsy, developmental delay, and severe intellectual disability. This gene plays a crucial role in the process of protein synthesis, and when it is altered, it causes cellular malfunction, which manifests itself in significant morphological changes and the induction of apoptosis. These alterations largely explain the clinical manifestations observed in affected patients. The objective of this study is to present the clinical case of a patient with a mutation in the EEF1A2 gene, with the aim of contributing to a better understanding of the syndrome and highlighting the importance of early diagnosis. The patient is a 37-year-old woman with downward slanting palpebral fissures, deep-set eyes, psychomotor developmental delay, severe intellectual disability, sleep disorders, autistic behavior, aggressive behavior, and seizures. A clinical exome study was performed, which identified the c.808G>A variant. This variant, which had initially been classified as of uncertain significance, was subsequently reevaluated and considered "probably pathogenic," as there was a direct correlation between the molecular findings and the clinical phenotype. Comparative analysis with previously published cases in the literature revealed that the patient's phenotype consistently matched the characteristics already described, including difficult-to-control epilepsy, severe developmental delay, and facial dysmorphia. In conclusion, the syndrome associated with mutations in EEF1A2 should be considered a rare clinical entity, but one with a characteristic phenotype. Molecular characterization is essential for an accurate diagnosis, thus enabling comprehensive patient management and timely genetic counseling.

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CR 8. *The role of BRCA2 in genetic integrity and its relationship with breast cancer*

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BRCA2 is a tumor suppressor gene crucial for DNA repair. A pathogenic variant of this gene causes hereditary breast and ovarian cancer syndrome, increasing the risk of developing breast cancer by up to 85%, and also increasing the risk of pancreatic cancer and melanoma. These variants are usually associated with estrogen receptor-positive and HER2-negative breast cancer, although any subtype can be present. A 42-year-old woman was evaluated due to a family history of cancer, which included a sister with a known pathogenic variant in the BRCA2 gene and another sister with breast cancer. The patient presented with precursor symptoms: mastalgia, menstrual irregularities, and palpable axillary lymphadenopathy since adolescence, which prompted a surveillance regimen. Despite having benign breast findings (BI-RADS 2), her risk profile warranted genetic testing, which revealed a pathogenic variant in BRCA2: c.8168A>G in a heterozygous state. A crucial factor in the risk of developing breast cancer due to BRCA2 gene variants lies in their epigenetic relationships and gene expression. For example, epigenetic silencing of the BRCA1/2 gene in the tumor, in the absence of a germline mutation, can lead to the "BRCAness" phenotype. This state causes the tumor to behave similarly to one with a BRCA mutation, affecting DNA repair pathways. These characteristics influence cancer susceptibility and response to treatments such as poly ADP-ribose polymerase inhibitors and chemotherapy. A surveillance plan was established with follow-up by oncology, gynecology, and dermatology for the detection of neoplasms. This case demonstrates how identifying a genetic predisposition, even in the absence of malignancy, is fundamental for implementing personalized risk reduction and prevention strategies.

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IP 01. *Impact of the Gut Microbiota as an Internal Exposome on the Modulation of the Histone Code: A Systematic Review*

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The internal exposome includes signals such as metabolites, hormones, and inflammation that interact with the genome and influence health and disease. Within this framework, the histone code—post-translational modifications regulating chromatin structure and gene expression—emerges as a key integration point. The gut microbiota connects these processes through epigenetic metabolites, particularly short-chain fatty acids (SCFAs) such as butyrate. A systematic review was conducted following PRISMA guidelines. Articles published between 2020 and 2025 were searched in PubMed and EuropePMC that assessed gut microbiota, SCFAs, and histone modifications. Of 100 initial references, 29 met inclusion criteria.

SCFAs showed consistent effects, mainly through histone acetylation at H3K9ac, H3K27ac, and H4K12ac, with loss of repressive marks such as H3K27me3. In immunity, they promoted acetylation in Il22 and Bcl6, strengthening the mucosal barrier and suppressing autoimmunity. In type 1 diabetes, autism, and chronic pain, dysbiosis reduced butyrate-producing bacteria and disrupted these signatures, changes reversed by microbiota transplantation or SCFA supplementation. In cancer, butyrate inhibited HDACs and activated tumor suppressor genes (SOCS1, CXCL11), enhancing immune infiltration and limiting tumor progression. In neuroscience, SCFAs regulate neurotransmitters and synaptic plasticity through histone acetylation in Tph2 and CRHR2. Nine studies were performed in humans (patients with type 1 diabetes, autism, colorectal, gastric, and breast cancer, hepatocellular carcinoma, and biliary atresia), while the remainder employed murine, porcine, or zebrafish models, confirming the molecular mechanisms. Current evidence supports that the gut microbiota, through butyrate and other SCFAs, modulates the histone code as an endogenous epigenetic regulator. These findings open therapeutic opportunities in immunity, cancer, metabolism, and neuroscience, although further clinical validation is required to translate them into precision medicine.

IP 02. *Characteristics and Applications of new RNA Therapies*

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Previous studies have demonstrated that the use of RNA molecules enables the modulation of biological pathways for the treatment of diseases, particularly those of genetic origin. RNA therapy acts by regulating post-transcriptional gene expression, making it possible to silence or enhance the production of a protein without altering the genome. RNA therapies represent a major innovation in the treatment of diverse diseases owing to their ability to intervene in gene expression. Nevertheless, their clinical application faces significant challenges, including RNA instability within the organism, difficulties in achieving targeted delivery to specific cells, and potential adverse reactions. These limitations underscore the need for extensive research to optimize both the safety and the efficacy of RNA therapies, thereby facilitating their clinical implementation. The aim of this review is to present selected examples of RNA-based gene therapies. Development: Several therapies have already yielded highly promising results. Notable examples include mRNA vaccines against COVID-19; therapies for propionic acidemia, in which RNA enables the synthesis of missing enzymes; RNA interference approaches; and antisense oligonucleotides designed to silence deleterious genes or block pathological proteins. Additional studies highlight the use of lipid nanoparticles and chemical modifications as delivery strategies. Collectively, these advances represent a novel pathway toward personalized and precision medicine. RNA therapies constitute an innovative alternative to conventional treatments. By acting prior to protein synthesis, they provide a means to address a wide spectrum of diseases with greater precision and therapeutic efficacy. To be established as a key tool in twenty-first-century medicine, further research and development are required to optimize their functionality and ensure their biological safety.

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IP 03. *Invisible marks in the epigenome: DNA methylation detection techniques in response to environmental pollutants*

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DNA methylation is an epigenetic modification that regulates gene expression and can be modulated by environmental *exposure. Several contaminants, including heavy metals, endocrine-disrupting chemicals, and particulate matter, have been shown to induce changes in methylation patterns, contributing to the onset of chronic diseases, developmental disorders, and cancer. Recent advances have led to increasingly sensitive and high-resolution methods for profiling DNA methylation. Among the most commonly used techniques are: (a) bisulfite sequencing, (b) methylation-sensitive PCR (MSP), (c) MeDIP-seq (Methylated DNA Immunoprecipitation Sequencing), and (d) methylation arrays such as the Illumina EPIC platform. These approaches have been widely applied to investigate the epigenetic impact of environmental contaminants. For instance, exposure to particulate matter (PM), black carbon (BC), ozone (O3), nitrogen oxides (NO2), and polycyclic aromatic hydrocarbons (PAHs) has been associated with global hypomethylation and genespecific changes linked to embryonic development, inflammation, and carcinogenesis. Insights into pollutant-induced DNA methylation changes offer valuable information on specific epigenetic signatures that may serve as sensitive biomarkers of environmental exposure and disease risk. These findings are driving the development of public health research strategies grounded in epigenetic science. In this context, the aim of the present study is to provide an up-to-date overview of the main techniques used for DNA methylation analysis, along with a synthesis of the scientific evidence in which these tools have been applied to assess pollutant-induced epigenetic alterations across diverse biological models.*

IP 04. *Air Pollution During Pregnancy: Immunological Alterations and Consequences in the Maternal-Fetal System*

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Air pollution represents a critical global public health concern. During pregnancy, both maternal and fetal compartments exhibit heightened susceptibility owing to immune adaptations and accelerated cellular growth. Atmospheric pollutants such as $PM_{2.5}$, NO_2, O_3, and CO can traverse biological barriers (including the placenta) thereby inducing systemic inflammation and epigenetic reprogramming. These processes have been associated with adverse obstetric outcomes and long-term sequelae in offspring, including neurodevelopmental impairments, obesity, and metabolic disorders. To synthesize current evidence regarding the impact of prenatal exposure to ambient air pollutants on immunological pathways and its implications for fetal developmental programming. A narrative literature review was performed, encompassing epidemiological, clinical, and experimental studies published over the past decade in international databases. Eligible articles evaluated prenatal exposure to air pollutants in relation to perinatal and developmental outcomes. Gestational exposure to ambient pollutants disrupts maternal immune homeostasis, impairs placental functionality, and adversely affects fetal health, with repercussions extending across the life course. These findings provide empirical support for the DOHaD Developmental Origins of Health and Disease) paradigm and underscore the urgent need for preventive public health policies, maternal-fetal medicine strategies, and integrative interventions to mitigate the impact of air pollution on maternal and child health.

IP 05. *Childhood Autism in Puebla: Epigenetic Factors and Social Repercussions.*

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Autism Spectrum Disorder (ASD) is a neurodevelopmental condition that affects social communication, behavioral flexibility, and sensory processing. Its prevalence has increased in recent years, reflecting both improved diagnostic practices and advances in research. ASD is now understood as a multifactorial condition in which genetic, environmental, and epigenetic factors interact. Epigenetics plays a key role by regulating gene expression during brain development, and it has been shown that prenatal infections, exposure to contaminants, nutritional deficiencies, or the use of specific medications may induce molecular modifications that increase the risk of autism. Diagnosis is based on criteria such as

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persistent difficulties in social interaction, repetitive behaviors, and early onset of symptoms. Screening tools such as the M-CHAT and ADOS enable early detection, while developmental red flags—including absence of babbling, reduced eye contact, or delayed language—allow recognition at critical stages. According to the DSM-5, ASD is classified into three levels of severity, defined by the degree of support required: from level 1, where challenges can be managed with structured assistance, to level 3, where individuals require intensive and continuous support across daily activities. In the educational field, recent literature highlights the urgent need to strengthen teacher training in theoretical, pedagogical, and attitudinal competencies to effectively address the diversity within the spectrum. The creation of structured learning environments, the use of predictable routines, and the reinforcement of collaborative work between schools, families, and communities are emphasized. Furthermore, public policies must be translated into concrete resources and inclusive strategies that guarantee equity and educational quality. In conclusion, ASD cannot be attributed to a single cause but rather to the complex interaction between biology and environment. A comprehensive understanding of its multifactorial nature opens new opportunities for prevention, early detection, and the development of inclusive and personalized interventions.

IP 06. *Domestic exposure to endocrine disruptors as a risk to hormonal balance and women's health*

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Everyday cleaning products contain chemical compounds that, while facilitating hygiene and disinfection, represent a significant source of exposure to endocrine disruptors (EDs). Among the most studied are dioxins, bisphenol A, phthalates, perchlorate, perfluoroalkyl substances (PFAs), phytoestrogens, polybrominated diphenyl ethers (PBDEs), polychlorinated biphenyls (PCBs), triclosan, atrazine, lead, arsenic, mercury, organophosphate pesticides, and glycol ethers. These substances can alter the synthesis, metabolism, and action of hormones, particularly affecting the female endocrine system. This review aims to provide updated information on ED exposure from cleaning products and their hormonal effects on women. This work was conducted in accordance with PRISMA guidelines and is based on original studies and systematic reviews sourced from PubMed, SciELO, and ScienceDirect. Current evidence shows that chronic exposure to these compounds can interfere with the hypothalamic-pituitary-ovarian-thyroid axis, causing menstrual disorders, thyroid dysfunction, infertility, and increased susceptibility to hormone-dependent conditions such as endometriosis and breast cancer. Certain biomarkers have detected the presence of EDs in blood, urine, breast milk, and reproductive tissues, confirming their bioaccumulation in these tissues. Furthermore, some compounds are capable of directly interacting with hormone receptors, regulating transcription factors, and modifying gene expression. During pregnancy, ED exposure compromises both maternal health and fetal neurodevelopment, particularly due to its impact on the thyroid axis. It has been suggested that some effects may be transmitted transgenerationally through epigenetic mechanisms. Exposure to EDs found in cleaning products contributes to a public health issue with gender biases, as women are more exposed due to biological factors and social roles. In Mexico, the lack of regulation on EDs and limited awareness of their risks demand coordinated action from authorities and social sectors to control their use.

IP 07. *Matrix Metalloproteinase 8 (MMP-8) as a Diagnostic Adjunct in Obstetric Infection by β-Hemolytic Streptococcus*

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During pregnancy, the fetus is enveloped by specialized membranes responsible for multiple essential functions. Prior to and during labor, these membranes rupture due to uterine contractions and the enzymatic activity of specific metalloproteinases.
A broad spectrum of metalloproteinases (MMPs) has been identified; however, in obstetric contexts, three are of particular relevance: MMP-2, MMP-8, and MMP-9. This discussion focuses on MMP-8, given its strong association with infectious processes—most notably those caused by β-hemolytic Streptococcus. In conjunction with tumor necrosis factor alpha (TNF-α), MMP-8 participates in the activation of the Fetal Inflammatory Response Syndrome (FIRS). During intra-amniotic infection, multiple pathways are triggered, including the activation of MMP-8 by TNF-α through the FIRS cascade. The interplay of these systemic and cellular mechanisms contributes to premature rupture of membranes (PROM), a condition with significant maternal and neonatal implications. The aim of this proposal is to explore the diagnostic value of MMP-8 and TNF-α through vaginal fluid sampling, thereby avoiding the risks and discomfort associated with amniocentesis, while enabling earlier detection compared with conventional histological examination of the placenta.

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IP 08. *Nutrition and Epigenetics: the social impact on health programming*

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In recent years, it has become clear that food not only provides energy but also acts as a signal capable of leaving traces in our genome. These epigenetic traces, modulated by diet and social conditions, can increase or decrease the risk of chronic diseases. Factors such as poverty, stress, or limited access to healthy food result in biological changes that explain why certain groups are more vulnerable to obesity, diabetes, or immunological disorders. Our objective is to present the available knowledge about the relationship between nutrition, social determinants of health, and epigenetic mechanisms, highlighting their impact on metabolic, immunological, and chronic diseases. A search was conducted for articles published between 2011 and 2023 that address diet, epigenetics, and health. Reviews and studies in humans and animal models focusing on obesity, diabetes, immunity, and early nutrition were primarily considered. The literature shows that maternal nutritional status and diet quality during the first 1,000 days of life influence the epigenome of children and grandchildren. High-fat or low-protein diets, as well as deficiencies or excesses of micronutrients such as folate, modify genes linked to metabolism and immunity. Phenomena of "metabolic memory" have been documented in diabetes, intergenerational transmission of obesity, and the positive impact of breastfeeding on epigenetic regulation. These findings confirm that social determinants, by influencing diet quality, amplify health inequalities through heritable markers. Nutritional epigenetics shows how social and dietary experiences are inscribed in our genes without altering their sequence. Understanding these processes opens the door to preventive interventions, from personalized nutrition to public policies that reduce health inequity.

IP 09. *Familial adenomatous polyposis progressing to tubular adenocarcinoma: case report with genetic approach*

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Familial adenomatous polyposis (FAP) is a rare autosomal dominant disorder caused by mutations in the APC tumor suppressor gene, leading to the development of numerous colorectal polyps. It entails a high risk of dysplasia and malignant transformation, particularly in the presence of KRAS oncogene mutations. Between 2 and 4% of early-onset colorectal cancer (EOCRC) cases arise from FAP, and the absence of treatment could significantly reduce life expectancy. Management strategies include prophylactic colectomy, endoscopic surveillance, and, in advanced stages, chemotherapy. 29-year-old female with rectal bleeding since adolescence, without medical attention due to socioeconomic limitations. In 2021, FAP was diagnosed by endoscopy, and after an interrupted follow-up due to pregnancy, a tubular adenocarcinoma of the right colon stage I, was confirmed in 2023 by histopathology preceding total proctocolectomy with terminal ileostomy. Additional molecular studies confirmed a KRAS mutation c.35G>A p.(G12Asp), ruling out anti-EGFR therapy. Analysis of this case highlights that molecular heterogeneity influences therapeutic response in EOCRC. Anti-EGFR agents are ineffective in tumors harboring KRAS mutations or originating in the right colon. Alternatively, anti-VEGF agents such as bevacizumab, aflibercept, and ramucirumab represent treatment options in challenging cases, although surgery remains the standard of care to ensure oncological control. This case emphasizes the relevance of personalized medicine, incorporating molecular and social determinants in clinical decision-making. It constitutes a valuable formative experience by linking genetics with clinical practice and integral patient care.

IP 10. *Mutations associated with lung cancer in non-smokers*

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Lung cancer in never-smokers (LCINS) is defined as a malignant neoplasm that develops in individuals who have never actively smoked tobacco, representing approximately 25% of all lung cancer cases worldwide. The emergence of LCINS highlights the influence of environmental, genetic, and endogenous factors that are still poorly understood. This presents a medical challenge for diagnosis and treatment, as it is often detected in advanced stages and exhibits distinct molecular profiles. Furthermore, it underscores the need to strengthen public policies related to air quality, regulation

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of environmental pollutants, and prevention of toxic exposures. The objective of this review is to present some of the mutations associated with the development of LCINS. The Sherlock-Lung project identified that the most frequent driver mutations in LCINS occur in EGFR, TP53, and KRAS. By geographical region, EGFR and TP53 mutations are predominant in East Asia, whereas KRAS mutations are more common in North America and Europe. Likewise, it has been observed that air pollution, especially exposure to fine particulate matter (PM2.5), is associated with an increased mutational burden and telomere shortening, suggesting an effect on carcinogenesis. Another mutation linked to LCINS is SBS40a, which is highly prevalent and has been associated with exposure to aristolochic acid in Taiwan. These advancements allow for a better understanding of the molecular heterogeneity of LCINS and highlight the importance of considering environmental, geographical, and genetic factors in its approach. Genes such as EGFR, TP53, KRAS, and SBS40a play a crucial role in its pathogenesis, offering opportunities to design prevention strategies and precision treatments that improve the survival and quality of life for patients with LCINS.

IP 11. *Importance of using non-invasive prenatal screening through fetal DNA analysis*

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Fetal genomic sequencing is a diagnostic tool that has proven to be very useful in prenatal settings. Its use allows for the identification of genetic abnormalities in fetuses. This technology will help facilitate early diagnosis, informed reproductive decisions, and improved neonatal outcomes. Fetal genomic sequencing allows the detection of treatable genetic diseases before birth. The lack of guidelines limits its clinical utility and creates uncertainty for physicians and families. Identifying childhood-onset disorders early would facilitate interventions that reduce neonatal morbidity and mortality, as well as improve reproductive planning and social preparation for parents. Therefore, it is necessary to establish clear criteria to guide the reporting of these findings, balancing medical benefit with respect for family decisions. This research has identified 296 genetic disorders, which can be treated in the prenatal or neonatal stages. Its development considered criteria such as the possibility of intervention before birth, the application of immediate treatments in the newborn, and the improved prognosis with early diagnosis. Some of the diseases considered include certain heart diseases and digestive disorders that can be stabilized with specific therapies. This proposal aims to demonstrate how fetal DNA analysis can be integrated into noninvasive prenatal screening in the future. This could lead to more personalized prenatal medicine, although it faces some clinical, ethical, and social obstacles to its implementation. Having early detection tools is essential for initiating specific clinical interventions and promoting informed decision-making. Their use will contribute to a better understanding of these diseases and improve their treatment options.

IP 12. *Pharmacogenetics in Pediatric Oncology: Towards Systematic Implementation*

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Pharmacogenomics is the discipline that studies how genetic variations influence the response of patients to medications. In pediatric oncology, where treatments are prolonged and complex, this tool could become particularly relevant. The treatment of childhood cancer involves a delicate balance between therapeutic efficacy and minimizing adverse effects. Prolonged exposure to chemotherapeutics and supportive medications increases the risk of serious toxicities. The lack of systematic implementation of preemptive pharmacogenetic testing delays the opportunity to personalize treatments. This represents a medical, social, and economic challenge, since adverse events generate higher costs and longer hospitalizations. The objective of this work is to highlight the need for specific evidence in pediatric populations to ensure safer and more effective treatments. The Princess Máxima Center study evaluated 1151 children with different types of cancer treated between 2018 and 2023. Relevant genes (TPMT, NUDT15, DPYD, and CYP2D6) and 28 drugs recommended by international guidelines were analyzed. The results showed that 92% of patients had at least one genetic variant and that 16% would have required a dose adjustment or drug change. Pioneering programs such as PG4KDS at St. Jude Children's Research Hospital have demonstrated the feasibility of this approach. However, the need to adapt guidelines developed for adults to the pediatric context persists, considering variations in child metabolism. Preemptive pharmacogenetics has the potential to benefit pediatric cancer patients. Its integration into clinical practice is feasible, since in many cases genetic information is already obtained during oncological diagnosis.

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IP 13. *Dolly the Sheep: A Milestone in Modern Biotechnology and Its Implications*

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Somatic cloning is a biotechnological technique that makes it possible to create a genetically identical organism from an adult cell. Dolly the sheep, born in 1996, was the first mammal successfully cloned by this method. The success of Dolly's cloning spawned a revolution with far-reaching medical, social and ethical implications. In the medical field, it opened the door to pioneering research into regenerative medicine and the possibility of creating replacement tissues. Socially, it triggered an intense global debate on the ethical limits of genetic manipulation, human cloning and animal welfare. Its legacy raises fundamental questions about the direction and control of biotechnology research. The development of Dolly was led by Ian Wilmut and his team at the Roslin Institute in Scotland, using the Somatic Cell Nuclear Transfer (SCNT) technique. This process consisted of removing the nucleus from an adult Finn Dorset sheep stem cell, inserting it into an enucleated oocyte. The cloned embryo is implanted in the womb of a surrogate mother. The significance of this achievement is that it refuted the scientific belief that differentiated somatic cells were unable to generate a complete organism. This breakthrough boosted stem cell research, laid the foundations for projects to conserve endangered species and create transgenic animals producing therapeutic proteins. Cloning is a global bioethical debate; regulatory frameworks have been established for the responsible use of cloning and associated genetic technologies. In the future, the principles demonstrated by Dolly will continue to be the basis for the development of advanced therapies of personalized and regenerative medicine.

IP 14. *Geneagent: Self-verifying language Agent for analysis of gene setts using domain databases*

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Gene set analysis seeks to identify the underlying biological mechanisms of gene groups with shared functions. Large language models (LLMs) can generate functional descriptions, but they often produce hallucinations, i.e., factually incorrect information. GeneAgent is an LLM-based AI agent that reduces these inaccuracies through a self-verification process. The main difficulty with LLMs is the generation of hallucinations, false content, compromising the reliability and interpretability of gene functions. Manual verification of these results is inefficient. The need for this analysis arises because some gene groups do not fully match known biological functions, but only share a part of them; in these cases, the available methods do not offer an adequate interpretation, making it essential to develop more accurate approaches. Development: GeneAgent overcomes this with an autonomous self-verification mechanism that interacts with specialized biological databases. Its four-stage pipeline (generation, self-verification, modification, and summary) validates statements by comparing them against curated knowledge from 18 databases, significantly reducing hallucinations. Evaluated on 1,106 gene sets, GeneAgent outperformed GPT-4 in accuracy and semantic similarity, offering more relevant and novel functional descriptions in real-world cases (e.g., mouse melanoma), facilitating knowledge discovery. GeneAgent generates more relevant and comprehensive functional descriptions, accelerating the discovery of biological knowledge and outperforming other LLMs. Outlook: Future explorations include integrating other LLMs, using additional domain databases, or optimizing modification prompts to further improve its effectiveness.

IP 15. *Use of CRISPR-cas9 in Modulation of Inteleukin 1 expression for therapeutic use*

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The function of immune responses is regulated by gene methylation. Interestingly, genetic research has developed new technologies such as CRISPR-Cas9, which has enabled the modification of genes. Altered immune responses are a significant problem in the development of cancer, as well as autoimmune diseases, where the expression of Interleukins (ILs) participates in the development of these pathologies. The use of CRISPR-Cas9 technology has allowed for the modification of gene expression, and this presents an opportunity to understand and work on cellular responses. The

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objective of this work is to provide evidence of gene expression modification using CRISPR-Cas9. The modification of the specific response of IL-1 was studied using the CRISPR tool in a myeloid lineage derived from B cells. It was found that the methylation of the IL-1 promoter regions could modify the expression of responses depending on the methylation of these areas. Consequently, it has been shown that it was possible not only to increase their expression by demethylation, but also to decrease it by hypermethylation. The hypermethylation of these genes not only induced the cell's inability to differentiate into the myeloid lineage but also increased its phagocytosis capacity and its activity in general inflammatory responses. Although this technique represents a therapeutic window in vitro, the complexity of the techniques and the absolute control required for experimentation in cellular and animal models still pose challenges. However, genetic modification will be a true bridge and a less ambiguous area on the path toward future therapies.

IP16. *Impact of Iron Deficiency During Fetal Gonadal Development*

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Iron deficiency (iron-deficiency anemia) is one of the leading nutritional problems in pregnant women, with a global incidence of up to 40%. Iron deficiency anemia has been associated with fetal neurological development failures, premature birth, and low birth weight. Recent studies suggest that iron deficiency may impair the function of a key epigenetic regulator involved in embryonic sex determination, proposing a direct link between iron metabolism and sex determination in mammals. Maternal iron deficiency reduces the enzymatic activity of the demethylase KDM3A. This enzyme is critical for the expression of the Sry gene in gonadal somatic cells, and its disruption can result in a development of a female phenotype in XY embryos. The objective of this work is to highlight evidence for the role of iron in gonadal development using a murine model. Maternal iron deficiency in mice blocks the activation of the Sry gene, which is required for testis formation. Consequently, XY embryos exhibited sex reversal with the development of female characteristics. This finding challenges the classical view that sexual differentiation is solely determined by chromosomal factors. The results demonstrate that nutrients such as iron modulate epigenetic processes during development. Specifically, iron is essential for activity of enzymes that release the Sry gene from repressive signals. Given the high prevalence of iron deficiency, these findings have broad public health implications. Thus, iron emerges as a critical regulator of fetal sex determination. Alterations in the uterine environment, such as iron availability, have the potential to influence embryonic development. This raises the possibility that iron deficiency in pregnant women could affect sexual differentiation in humans, particularly in individuals with genetic predisposition or in populations with a high incidence of iron-deficiency anemia.

IP 17. *New classification of burkitt lymphoma types based on their epigenetic characteristics*

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Burkitt lymphoma (BL) is the most prevalent type of non-Hodgkin lymphoma (NHL) of B cells in children, representing 50% of pediatric NHL cases, and is rare in adults. BL is classified according to its clinical variants (endemic or sporadic) and the patient's age (pediatric or adult). One of the causal agents associated with BL is the Epstein-Barr virus (EBV-positive); however, there are other etiologies (EBV-negative). Based on the types of associated mutations, four subgroups have been proposed, so the identification of these epigenetic patterns could lead to a more precise risk assessment for patients with Burkitt lymphoma. In an epigenetic study of genomic DNA from 218 patients with Burkitt lymphoma. The genomic characteristics were identified based on their degree of methylation (hypo or hyper), thus are referred to as the HypoBL or HyperBL epitypes. The HyperBL epitype showed a strong association with mutations linked to EBV-positive cases, highlighting its importance in the pathogenesis of BL, while HypoBL showed a more balanced distribution between EBV-positive and EBV-negative tumors. In adult patients with HyperBL, there is greater tumor progression and significantly shorter overall survival compared to HypoBL. Regarding their cellular characteristics, HyperBL is epigenetically similar to centroblasts, whereas HypoBL is more similar to memory B cells. Therefore, the classification of these epitopes proved to be more relevant than EBV status in explaining variations in mutational burden. The epigenetic identification of HyperBL and HypoBL in Burkitt lymphoma is strongly associated with clinical outcomes and related mutations, compared to EBV-positive or EBV-negative classification. Thus, the use of demethylating agents could be part of future treatment strategies.

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IP 18. *Placental genes that are expressed differently according to sex during fetal development and their implications*

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The placenta is the fundamental organ of fetal development. Its functionality depends on the genes it expresses during fetal development and the maintenance of pregnancy. There is clinical evidence of the association between male or female fetuses and certain genetic pathological events. Therefore, the importance of this research lies in its potential to identify diseases in intrauterine and extrauterine life. These epigenetic differences may help explain why the mother can develop preeclampsia when carrying a male fetus. Such investigations could contribute to the prevention of various early fetal developmental disorders. The objective of this work is to present evidence of differential gene expression and its clinical association. This study identified 6,077 sex-differentially methylated sites (sex-DM), with a higher probability of male hypermethylation. It also identified 1,839 sex-biased methylation quantitative trait loci (sex-mQTL), and 13 sex-biased expression quantitative trait loci (sex-eQTL), many of which showed sex-specific effects. Sex-dependent methylation clusters in CpG loci affect gene transcription levels, being more common in the promoter region and less so at the transcription start site. An example in female fetuses is the methylation of the KCNQ1OT1/CDKN1C gene, implicated in Beckwith-Wiedemann syndrome. In male fetuses, effects were observed in a set of hemostasis/coagulation-specific genes, suggesting a possible epigenetic explanation for the male predominance in pregnancy-related pathologies. These findings highlight the potential role of placenta-mediated sex differences in physiological developmental traits and in diseases throughout life.

IP 19. *Impact of Fructose consumption during pregnancy and the risk of heart defects in offspring*

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Excessive fructose intake has been linked to metabolic and cardiovascular disorders such as obesity, type 2 diabetes mellitus, and cardiac hypertrophy. The latter refers to the abnormal enlargement of the myocardium due to an increase in the size of cardiomyocytes. According to INEGI 2024, cardiovascular diseases are the leading cause of death in Mexico, followed by diabetes. High fructose consumption during pregnancy may interfere with fetal cardiovascular development. It is not yet clear whether it is associated with cardiac hypertrophy in the fetus, which could increase the risk of heart disease later in life. Various studies have shown that fructose can induce oxidative stress and disrupt the HIF-1α pathway, promoting pathological cardiac hypertrophy. In animal models, fructose intake during pregnancy results in myocardial hypertrophy in both the mother and offspring, even after postnatal exposure is discontinued. It has been proposed that fetal programming, mediated by epigenetic mechanisms, perpetuates these adverse effects on adult cardiac function. Furthermore, the combination of fructose with other stress conditions enhances cardiac remodeling and fibrosis. Antioxidant treatments such as resveratrol or SkQ1 have shown potential to reduce oxidative damage and attenuate hypertrophy. On a metabolic level, fructose not only affects the liver but also serves as a direct energy source for the heart under stress, promoting cardiac muscle growth. Maternal exposure to fructose is associated with changes in HIF-1α expression and osmolarity, which predispose offspring to cardiac hypertrophy. Intake during pregnancy worsens cardiac damage by increasing oxidative stress, reducing glutamine protection, and decreasing autophagy. The importance of reducing maternal fructose consumption emphasizes the prevention of cardiovascular risks in future generations.

IP 20. Uso de Inteligencia Artificial para personalizar la Inmunoterapia en cáncer

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Lung cancer is a proliferation of lung cells that have undergone DNA mutations. This type of cancer is the leading cause of mortality worldwide, representing 18.4% of all cancer deaths globally. Lung cancer is often detected late and has a low survival rate. It also places a heavy burden on healthcare systems, requiring costly and prolonged treatments.

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Socially, it has a significant impact, affecting the quality of life of patients and their families, and causing emotional and work-related issues. For all these reasons, lung cancer is a major health problem that requires better strategies for diagnosis and treatment. The implementation of liquid biopsy makes it possible to detect epigenetic biomarkers in the blood in a non-invasive manner. This will help identify patients who will respond to immunotherapy and those who will present resistance. In addition, the use of artificial intelligence applied to the analysis of data from 250 patients with advanced non-small cell lung cancer made it possible to optimize therapeutic decisions. Another highlight is the acquisition of European and national funding, which ensures the sustained development of research. Taken together, these achievements represent a significant step forward toward precision medicine, improving patients' expectations and quality of life. This research seeks to decipher the keys to making immunotherapy more personalized and effective, with a clear focus on improving the quality of life of lung cancer patients. This indicates a move toward therapies that are better adapted to the individual characteristics of each patient, rather than just standard treatments.

IP 21. *Mechanism, senescence and persistence: a new perspective in cancer type identification*

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Cancer continues to be one of the leading causes of morbidity and mortality globally. In Mexico, the mortality rate was 70.8 deaths per 100,000 inhabitants. In the development of cancer, cells are persistent and senescent. This confers tolerance to drugs, and they are even capable of entering a state of reversible proliferative arrest and resuming growth once therapy is discontinued. They also exhibit specific epigenetic mechanisms, such as the repression of inflammatory genes mediated by the heterochromatic mark H4K20me3, which confers survival and immune evasion advantages. Our objective is to present the results of this research. Studies in tumors treated with chemotherapy showed that some intratumoral cells can experience senescence, while others may persist. Furthermore, treatments with the INK128 inhibitor (which blocks cell growth and metabolism) showed overexpression of biomarkers (DEC2 and p27), suggesting that cells develop persistence. Senescence is a state of low reversibility in which cells remain arrested for the long term. They possess senescence-associated secretory proteins (SASP), which include cytokines, chemokines, and matrix remodeling proteins. This exacerbates immunosuppression after chemotherapy. The identification of senescent-type tumors could be treated with the agent doxorubicin or the CDK4/6 inhibitor palbociclib, facilitating tumor relapse and promoting immunosuppression. Meanwhile, persistent tumors treated with inflammatory gene silencing techniques (cytokines and interferons) through heterochromatic H4K20me3 methylation of the promoter region blocked tumor progression. The eradication of cancer cells provides a new perspective on the multiple capabilities of these malignant cells. Understanding their mechanisms opens opportunities to study possible routes to eradicate them.

IP 22. *Importance of generation of epigenetic bases of hematological neoplasms*

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Hematological malignancies are characterized by the uncontrolled proliferation of malignant cells in the blood, bone marrow, or lymph nodes. In 2019, there were more than 1.3 million new cases worldwide. Hematological malignancies are a major health problem due to their high morbidity and mortality rates. The diversity of subtypes and diagnostic challenges delay the selection of appropriate treatment, negatively impacting patient survival and placing an emotional burden on families. The lack of accurate diagnosis tools and predictors of therapeutic response limits the individualized management of each patient. Our objective is to present results from databases created from cultured cancer cells. The Joseph Carreras Leukemia Research Institute recently published a database of cultured cancer cells. This database contains methylation profiles of different types of leukemia, lymphoma and myeloma in both humans and mice, designed for research and clinical applications. It also provides information on the sensitivity of more than 300 drugs across more than 200 cell lines. The study of this database will enable the development of algorithms that can predict resistance and sensitivity to specific drugs. The creation of epigenetic databases of malignant blood cells represents a valuable resource for improving diagnosis, classification, and clinical research or hematological malignancies. This advancement will contribute to increased survival, improved quality of life for patients, and the development of more personalized therapies.

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IP 23. *Identification of Epigenetic Mechanisms that Generate Changes in Cancer Cells*

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Cancer is a complex pathology involving cellular and molecular alterations. Cancer-related mortality has surpassed that of cardiovascular diseases for the first time. Cancer mortality could be reduced if detected early. The traditional approach to studying tumor cells was based exclusively on analyzing mutations in oncogenes and tumor suppressor genes. However, recent studies on the epigenetic properties of cancer have revealed its remarkable ability to adapt and survive. Several mechanisms have been proposed that highlight the risk of genomic instability and gene deregulation, making it essential to review these mechanisms as potential diagnostic tools. Six epigenetic hallmarks of cancer cells have been identified: a) reactivation of ancient viral sequences within our genome, b) silencing of tumor suppressor genes through DNA methylation, c) histone modifications regulating gene expression, d) remodeling of the three-dimensional structure of the cell nucleus, e) epigenetic instability that drives cancer evolution, f) a love-hate relationship with genetic alterations in cancer. Therefore, the application of emerging epigenetic technologies will be required to uncover new rules and patterns in cancer. Recognizing alterations that occur in cancer cells will help explain tumor heterogeneity, provide opportunities for improved diagnosis, and enable progress toward more personalized and effective cancer therapies.

IP 24. *Effect of Epigenetic and Chronological Age on Cardiovascular Health*

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In 2022, 19.8 million people died from cardiovascular diseases, representing approximately 32% of all global deaths, according to the WHO. Recently, epigenetic aging has been associated with this condition, a phenomenon in which a person's biological age does not match their chronological age. Understanding the factors involved in aging and disease onset contributes to the development of preventive strategies. The adoption of healthy lifestyle habits can delay epigenetic aging. Measuring epigenetic age through DNA methylation makes it possible to identify high-risk individuals with a biological age greater than their chronological age. Slowing epigenetic aging through balanced habits positively impacts the incidence of cardiovascular diseases and their consequences. Research has shown that aging is influenced by genetics, suggesting that various lifestyle factors can modify epigenetic effects. DNA methylation has been identified as a marker to estimate epigenetic age and predict the risk of age-related diseases. The Framingham Heart Study demonstrated the impact of gene expression related to cardiovascular diseases and its association with disease risk and greater longevity. Interestingly, some epigenetic modifications are reversible, which highlights epigenetics as a bridge between lifestyle and the prevention of chronic diseases. Slowing epigenetic aging through a healthy lifestyle is an early prevention strategy that broadly impacts multiple diseases. Promoting healthy aging may reduce costs associated with chronic treatments, hospitalizations, and long-term care.

IP 25. *Smoking Electronic Cigarettes Also Leaves a Mark on DNA*

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In Mexico, lung cancer accounts for 10.4% of cancer incidence. The National Health and Nutrition Survey (ENSANUT) reported that 2.6% of adolescents and 1.5% of adults use electronic cigarettes, in comparison with conventional cigarette smoking. Recent reports from ENSANUT have indicated not only an overall increase in e-cigarette use but also a marked rise among younger populations (10–19 years). Furthermore, the Federal Commission for Protection against Sanitary Risks in Mexico conducted a chemical analysis of e-cigarette devices and identified 30 undeclared substances in their contents. As a result, the Official Gazette of the Federation issued a decree prohibiting the commercialization and sale of e-cigarettes, which came into effect on January 17, 2025. Epigenomic studies on tobacco and e-cigarette exposure have identified 535 CpG sites with significant methylation alterations. These included two patterns of hy-

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pomethylation (epithelial and immune) and two distinct patterns of hypermethylation (proximal epithelial and distal epithelial). Notably, methylation signatures were detected in oral samples of individuals who subsequently developed lung cancer, in some cases up to 22 years prior to diagnosis, particularly among smokers. Moreover, e-cigarette users exhibited epigenetic footprints that were strikingly similar to those of conventional smokers. This evidence suggests that e-cigarette use is not biologically innocuous and may induce epigenetic alterations with pro-carcinogenic potential. Interestingly, in former smokers, these changes tend to partially revert; however, complete normalization is not achieved, resulting in persistent molecular damage. Although e-cigarettes were initially promoted as a harm-reduction strategy for conventional smoking, emerging evidence indicates that their use may represent a major risk factor for cancer development, particularly among younger populations.

IP 26. *Use of Liquid Biopsies in the Diagnosis of Advanced Cancer*

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Cancer is one of the world's leading causes of death, accounting for almost 10 million fatalities in 2020 and around 19.3 million new cases being reported annually. Early cancer diagnosis has the potential to save lives and significantly reduce treatment costs. However, current clinical tests lack sensitivity and specificity for early-stage cancers. Overall, cancer-related mortality is considerably higher in poorer countries, particularly among people under 65, and most of these deaths are related to a lack of timely diagnosis. Using epigenetic information obtained from blood samples of patients with advanced cancer can provide insight into disease progression and help select appropriate treatments. The use of liquid biopsies has identified several biomarkers of interest for clinical practice. This has been accompanied by studies investigating the activation of regulatory expression regions through DNA methylation and histone modifications in these regions. One study of 433 cancer patients identified the activation of gene promoters, such as MYC and EGFR, and the repression of tumour suppressor gene promoters. The study also determined the activity of genes coding for drug targets such as ERBB2, ERBB3, NECTIN4 and DLL3. Studying advanced cancers through direct tissue sampling demonstrates the prognostic potential of plasma epigenomic profiles. Studies focusing on the application of epigenetic information are key to making advances in the timely diagnosis of cancer. In the future, liquid biopsies may expand their scope and become fundamental to the global fight against cancer.

IP 27. *Epigenetic Changes in Sperm Associated With Paternal Age*

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Advanced paternal age is associated with reproductive risks and developmental alterations in offspring, primarily linked to epigenetic changes in sperm. In Europe, the age of fatherhood has risen over recent decades; currently, 95% of men aged 18 to 30 still haven't had children. This global trend of delayed fatherhood is a growing concern, raising new medical and social issues relevant to both reproductive and pediatric health. Understanding the underlying mechanisms of these effects is crucial for developing preventive measures and providing clinical guidance. Research suggests that epigenetic changes in sperm are a key factor in the transmission of these risks to future generations. These modifications, which don't alter the DNA sequence itself but can change how genes are expressed, are a significant area of study. An analysis of sperm samples from 73 men revealed that aging is linked to significant changes in DNA methylation. Specifically, 1,162 hypomethylated regions (where DNA is less methylated) and 403 hypermethylated regions (where it is more methylated) were identified as a man's age increased. A notable finding was a disproportionate number of these changes on chromosome 19. Interestingly, 241 of these regions were associated with biological processes crucial for development and the nervous system. This supports the hypothesis that epigenetic changes can influence offspring behavior and neurodevelopment, potentially explaining the increased risk of certain pathologies in children of older fathers. In conclusion, advanced paternal age correlates with specific epigenetic alterations in sperm, especially in regions vital for the nervous system. These changes may be a key mechanism behind the increased risk of reproductive and neurobiological disorders in the next generation. Further research into these specific epigenetic markers could lead to new diagnostic and therapeutic strategies.

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IP 28. *Early Diagnosis of Preeclampsia Through the Use of Liquid Biopsy and Epigenetics*

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Preeclampsia is characterized by elevated blood pressure in pregnant women. The development of preeclampsia is associated with increased fetal morbidity and mortality. It has an incidence of 2-8% pregnancies worldwide according to the WHO. Preeclampsia represents a public health problem with medical, social, and economic impact. Its complications include premature birth, fetal growth restriction, and long-term maternal cardiovascular risk. Current screening methods are complex and expensive, as they combine clinical, biochemical, and ultrasound markers, limiting their application in low-resource settings. This perpetuates inequity and hinders prevention. Therefore, it is essential to develop accessible, sensitive, and applicable tests from the first trimester that allow for the initiation of cost-effective preventive measures. Recently, the use of liquid biopsies, which allow the analysis of DNA methylation, began. On the other hand, it has been found that changes in the methylation of circulating fetal DNA (cfDNA) occur before and during the onset of preeclampsia. Based on this finding, a test capable of assessing the risk of preeclampsia from the first trimester of pregnancy was developed. To do this, they used liquid biopsies, which allow the analysis of DNA methylation in placental cells. These allow the analysis of the small proportion of free DNA derived from placental cells present in maternal blood. The validation of this predictive model that classifies pregnant women according to their risk level enables early detection and the timely implementation of preventive measures. cfDNA methylation analysis is an innovative and effective method for predicting early preeclampsia. Its integration into prenatal practice can optimize prevention and reduce maternal health inequities.

IP 29. *Epigenetic Profiles Related to the Severity of COVID-19*

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COVID-19 is caused by a virus belonging to the SARS-CoV-2 family. The Government of Mexico reported 7 million positive cases and 334,336 deaths. The clinical evolution of COVID-19 is highly heterogeneous, ranging from asymptomatic or mild cases to severe respiratory failure requiring intensive clinical support. There is an urgent need for epigenetic biomarkers that enable early stratification of the risk of severe progression. An epigenome-wide study (EWAS) identified 44 CpG sites whose blood methylation significantly distinguishes between patients with mild versus severe courses, including key genes such as AIM2 and HLA-C associated with the interferón response. This led to the development of the EPICOVID epigenomic signature with high predictive accuracy (90%). This tool could be integrated with clinical variables to improve patient management, anticipate appropriate interventions, and reduce the healthcare burden resulting from the most severe cases. Researchers at the Josep Carreras Institute and the Bellvitge Biomedical Research Institute studied epigenetic profiles through DNA methylation analysis in over 400 COVID-19 patients without comorbidities or previous risk factors. They compared asymptomatic, mild, and severe patients (hospitalized or requiring assisted breathing). They identified 44 distinct genomic methylation marks associated with disease severity. With this, EPICOVID was developed, achieving a predictive accuracy of 83.5%. This will allow for the early identification of patients at higher risk, the implementation of intensive monitoring and timely treatment, and the alleviation of pressure on the healthcare system. It is also noteworthy for its usefulness in settings with limited access to vaccines, allowing high-risk groups to be prioritized. Integrating epigenetic information into clinical prediction models can improve personalized medicine, enable earlier interventions, and help prevent healthcare system collapse.

IP 30. *Epigenetic Analysis to Predict the Risk of Developing Nephropathy in Diabetic Patients*

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Diabetic nephropathy is the glomerular sclerosis and fibrosis caused by the metabolic and hemodynamic changes of type 2 diabetes (T2D). In Mexico, the prevalence of patients with T2D is 53%. Diabetes has a history of being a metabolic disorder affecting more than 500 million people worldwide, while diabetic nephropathy has become one of its most serious complications. DNA methylation studies to predict risk in pathologies can be applied to diabetes and its main complication, kidney damage. Advances in epigenetics offer new alternatives for the early detection and prevention

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of nephropathy in diabetic patients. This study analyzed DNA methylation levels in more than a thousand Scandinavian and Asian patients. It was observed that low methylation in CTCF and POL2B binding regions is associated with an increased risk of kidney disease. Furthermore, it was found that these epigenetic changes alter the expression of genes such as MTOR, RPTOR, IRS2, TXNRD1, LCAT, SMPD3, and COL1A2. These genes are involved in key processes such as insulin signaling, lipid metabolism, and the development of fibrosis. The researchers emphasize that an epigenetic test would be more stable and less invasive than kidney biopsies. This tool could be applied in remote areas for early detection of the disease. The findings open the door to new diagnostic and preventive strategies for diabetic nephropathy. Advances in epigenetic biomarker research could provide us with another, more stable and less invasive standard for the early detection and management of renal complications in type 1 diabetes mellitus. It remains to be seen how cost-effective these tests are for their incorporation into medical practice.

IP 31. *Epigenetics in the Modulation of Aging*

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Diseases associated with aging such as Alzheimer's, osteoarthritis, and geriatric syndromes have increased their incidence. Recent studies in yeast models indicated that chromatin modifiers and histone alterations can modify the aging process. Loss of epigenetic regulation is one of the main causes of aging, as it alters chromatin organization and affects gene expression. It has been proposed that restoring epigenetic organizations has the potential to reverse aging. So, these modifications could prevent, treat or even reverse age-related diseases. However, this approach is still new and lacks evidence in humans. Our objective is to show the evidence of studies in murine models. Mutations have been identified that force chromatin-modifying proteins to move to damaged areas, altering the accessibility of genes. These epigenetic changes stemming from DNA repair are considered a primary cause of aging. Murine models with DNA damage reported epigenetic disorganization and signs of premature aging. However, after a treatment that delivers OSK genes (Oct4, Sox2 and Klf4) using viral vectors, their organs and tissues regained functionality, indicating that cells can be epigenetically "reprogrammed". This shows that biological age in complex organisms can be precisely controlled, advanced or delayed. Consequently, manipulating the epigenome not only accelerates or stops aging, but also opens the possibility of reversing it. Epigenetics has a promising future for intervening in the aging process and age-related diseases. Although the findings come from studies in animal models, new research is opening towards cell reprogramming through epigenetic therapies in humans.

IP 32. *Generation of Epigenetic Profiles as a Diagnostic Method*

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Multisystem Inflammatory Syndrome in Children (MIS-C), related to the SARS-CoV-2 infection, is characterized by a cytokine storm (IL-1β, IL-6) that triggers epithelial damage and inflammation, potentially leading to multiorgan failure. It affects 1–2 per 100,000 patients under 21 years of age who become infected. MIS-C represents one of the most challenging phenomena to emerge during the COVID-19 pandemic, given its intense inflammatory response and multiorgan involvement—manifestations not exclusive to SARS-CoV-2 infection. Similar features have also been observed in patients with Kawasaki disease, suggesting shared mechanisms. This is particularly relevant considering the increase in Kawasaki cases reported after the 2009 influenza pandemic. The frequent occurrence of gastrointestinal symptoms and cardiac involvement further underscores the need to understand its molecular basis. Genome-wide DNA methylation was analyzed in blood samples from 43 MIS-C patients, 15 COVID-19 patients without MIS-C, and 69 non-COVID-19 children. Thirty-three genomic sites associated with MIS-C development were identified, involving immune response regulators such as ZEB2, GPR111, SH2D1B, and CUL2. Based on these findings, a characteristic epigenetic profile (EPIMISC) was defined, linked to dysregulated cellular programming that promotes hyperinflammation and tissue damage. The potential use of epigenetic drugs is also suggested as a therapeutic strategy. The study identified the EPIMISC epigenetic signature associated with MIS-C, which alters key immune response genes and contributes to hyperinflammation. This makes it a strong candidate as a diagnostic and prognostic biomarker. Moreover, it opens the possibility of developing shared therapeutic strategies that modulate methylation and mitigate hyperinflammation in pediatric inflammatory syndromes.

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IP 33. *Participation of Gene Expression From Both Parents in the Placenta During Fetal Development*

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Intrauterine growth restriction, a pathological condition where the fetus does not reach the expected weight for its gestational age, occurs in 10% to 15% of pregnancies and is a major cause of perinatal morbidity and mortality. The placenta is a highly relevant temporary organ during gestation, as it acts as the physiological interface between the mother and the fetus. Its main function is to ensure the proper exchange of oxygen, nutrients, and hormones, which are essential for fetal growth and development. The imbalance in the fetus's nutrient uptake through the placenta is regulated by IGF2. Failures in this pathway can lead to metabolic complications and chronic degenerative diseases such as diabetes, hypertension, and obesity, which are prevalent in most of the Mexican population. This, in the long term, increases healthcare costs for intensive prenatal care and hospitalizations, posing a problem for communities that lack access to health services. As pregnancy progresses, the placenta undergoes a remarkable expansion of its vascular network, which is used for the fetus/placenta exchange. The regulation of this expansion is managed by the IGF2-IGF2R pathway, where paternal IGF2 stimulates placental angiogenesis, while maternal IGF2R modulates and controls this signaling. This provides a scientific basis for addressing pregnancy complications related to alterations in fetal growth and placental function. The interaction between the IGF2 and IGF2R genes is crucial for establishing a balance that allows for optimal fetal growth, which opens a gap for pharmacogenomics in the precise manipulation of nutrient supply mechanisms.

IP 34. *The Epigenetic Signatures of Chronic Pain*

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Chronic pain is defined as an unpleasant and painful sensation that persists for a prolonged period. Epigenetic marks or methylation is one of these epigenetic marks that adds methyl groups to specific genes to turn them off or silence them. Chronic pain is a public health issue. Diagnosing it through subjective questionnaires with low accuracy leads to ineffective treatments, chronic pain, and a greater social and economic burden. The prevalence of chronic pain varies from 16% to 70% of the population. Specific epigenetic marks for each type of pain could be the key to changing this situation, offering an opportunity to develop early diagnostic biomarkers. Stenz's study analyzed the epigenome of 57 patients (20 controls, 18 with nociceptive pain, and 19 with neuropathic pain). In patients with nociceptive pain, alterations in the methylation of 12 neuromusculoskeletal genes and 8 immune genes were detected, while in patients with neuropathic pain, changes were identified in 6 neuromusculoskeletal genes and in the DCAF15 gene, which is related to the immune response. A key finding was that methylation patterns were completely different between the two types of pain, demonstrating that specific epigenetic signatures exist. These results open to the possibility of developing diagnostic tests using blood analysis, which would be useful for objectively classifying chronic pain and improving patient follow-up. In the future, the inclusion of epigenetic analysis in clinical practice would be a great tool that could allow us to make diagnoses more accurate, thus enabling us to implement personalized treatments that are tailored to each patient's type of pain.

IP 35. *Epigenetic Mechanism That Regulates the Effect of Early Stress in Adult Life*

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Stress is a physiological reaction of the body to certain stimuli, involving the activation of the nucleus accumbens. In Mexico, 7/10 people experience stress. Low levels of stress help maintain alertness and avoid dangerous situations. Prolonged exposure affects physical and mental health. Early exposure to stress affects health and development because the brain is very sensitive to environmental stimuli during childhood. Chronic stress alters brain structures related to memory, emotional control, and the regulation of stress responses, causing hyperactivation of the hypothalamic-pitu-

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itary-adrenal axis and difficulties managing stress in adulthood. This is associated with an increased risk of psychiatric disorders and chronic diseases. Specific methylation of histone H3K79 in nucleus accumbens neurons has been reported to promote molecular and physiological changes. This modification reprograms D2 dopaminergic neurons, altering the response capacity to future stressors. This epigenetic change is mediated by enzymatic mechanisms, highlighting Dot1L (adds the modification) and Kdm2b (eliminates the modification). This pathway regulates the expression of flavonoids, compounds with antidepressant potential. In a murine model, overexpression of Dot1L (increased H3K79me2 signals) reproduces the effects of early stress on gene expression, while its inhibition significantly blocks these changes. The drug "pinometostat" reduces the H3K79me2 signal in the nucleus accumbens. These findings point to Dot1L as a promising therapeutic target against the adverse effects generated by stress. The discovery of an epigenetic mechanism involved in the response to early stress opens a field of research that should deepen the understanding of the interactions between environmental, molecular and genetic factors that determine vulnerability or resilience to stress.

IP 36. Aluminum- and Lead-Induced Epigenetic Alterations and Their Relationship with Parkinson's

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Parkinson's disease (PD) is a neurodegenerative disorder characterized by the progressive loss of dopaminergic neurons and the accumulation of α-synuclein. Exposure to toxic metals, such as lead (Pb) and aluminum (Al), has been associated with the appearance of various epigenetic modifications linked to the development of PD. The objective of this review was to analyze recent evidence on epigenetic alterations induced by Pb and Al exposure that promote neurodegeneration in PD.
A search was conducted in PubMed, Scopus, Web of Science, and the Cochrane Library using PRISMA criteria. The Boolean equation "Parkinson's" AND (heavy metals OR lead OR cadmium OR mercury) AND (epigenetic OR DNA methylation OR histone modification) was applied, including articles published between 2019 and 2025 in English and translations from other languages. Screening and selection were performed using the Rayyan tool. Of 195 studies identified, 23 met the inclusion criteria. The findings show that Pb exposure causes hypomethylation in genes related to oxidative stress and neuronal function, in addition to altering histone acetylation and microRNA expression, which favors dopaminergic dysfunction. Furthermore, Pb-induced activation of LINE-1 elements was observed, associated with PD progression. Regarding Al, it has been described to activate monoamine oxidase B, accelerating dopamine degradation and promoting α-synuclein aggregation, a characteristic phenomenon of the disease. Together, these metals exacerbate pathological epigenetic processes, enhancing neurodegeneration. Evidence supports that Pb and Al contribute to the development and progression of PD through epigenetic mechanisms. Delving deeper into these processes will allow us to establish preventive strategies, design more effective therapies, and explore the role of other toxic metals that are still understudied.

IP 37. *Breastfeeding as a Modulator of Epigenetic Methylation of Metabolic Genes FTO and LEP in Early Childhood*

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Exclusive breastfeeding during the first six months is associated with long-term metabolic and developmental benefits. Early nutrition influences growth programming and adipogenesis through epigenetic mechanisms. FTO and LEP genes regulate energy metabolism and fat accumulation, and their methylation patterns have been linked to increased risk of childhood obesity. In Mexico, only 33% of infants receive exclusive breastfeeding, while childhood obesity prevalence is high. Despite its epigenetic impact on metabolic genes, few studies link breastfeeding with FTO and LEP regulation in early childhood, limiting preventive strategies. Breastfeeding modulates the epigenetic programming of LEP and FTO, affecting early obesity. In LEP, breastfed infants show promoter hypermethylation, reducing expression and preventing hyperleptinemia and leptin resistance [6]. Exosomal microRNAs such as miR-26a regulate adipocyte differentiation, while miR-148a and miR-21 modulate epigenetic enzymes (DNMT1, TET2). In FTO, exclusive breastfeeding mitigates the effect of the risk allele rs9939609-A through epigenetic changes in intronic regions interacting with IRX3/IRX5. The first 1000 days are a critical window for interventions that reprogram growth and metabolism via epigenetic mechanisms. Exclusive breastfeeding acts as an epigenetic modulator of LEP and FTO, reducing obesity

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risk through promoter hypermethylation and exosomal microRNAs. Effects are particularly relevant in carriers of risk alleles, modulating growth and adiposity. Breastfeeding is therefore a key early-life strategy for obesity prevention and healthy metabolic programming.

IP 38. *The Epigenetic Landscape of Post-Traumatic Stress Disorder: A Scoping Review of DNA Methylation*

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Post-traumatic stress disorder (PTSD) represents a severe condition triggered by exposure to high-conflict events, characterized by a range of symptoms including re-experiencing, avoidance, and hyperarousal. Epigenetics, particularly DNA methylation, is a mechanism involved in mediating the traumatic experience and gene expression, aiming to explain individual variations in vulnerability, resilience, and treatment response. This epigenetic modification represents an important advance in understanding the relationship between contextual factors and the pathophysiology of PTSD. Despite the growing volume of research on the epigenetics of PTSD, there remains a critical need to synthesize and map the existing evidence regarding specific DNA methylation patterns associated with this disorder. It is necessary to identify the most consistently altered genes and biological pathways, as well as to highlight future opportunities for translational research in this field. A scoping review was conducted following the PRISMA-ScR guidelines. The search strategy implemented Boolean operators in the PubMed and Scopus databases, identifying 626 potentially relevant articles. After removing 339 duplicates, 287 articles were screened using PCC criteria (Population: humans diagnosed with PTSD; Concept: DNA methylation; Context: studies on epigenetic biomarkers). Full-text evaluation with predefined inclusion and exclusion criteria resulted in the final selection of 51 articles for data extraction. Analysis of the evidence reveals that alterations in DNA methylation, particularly in genes regulating the hypothalamic-pituitary-adrenal (HPA) axis such as NR3C1 and FKBP5, play a crucial role in the pathophysiology of PTSD. A hypomethylation pattern is associated with greater vulnerability and symptom severity. Additionally, emerging findings highlight the acceleration of epigenetic aging (GrimAge) and mechanisms of intergenerational transmission of trauma-related epigenetic marks, suggesting both clinical and transgenerational implications. This review highlights the role of DNA methylation, particularly in HPA axis-related genes, in mediating the effects and development of PTSD. The findings support the potential of epigenetic biomarkers for early identification of vulnerability, prediction of therapeutic response, and the development of targeted interventions. Future research may focus on validating clinical findings, exploring mechanisms of epigenetic reversibility, examining transgenerational factors, and translating these discoveries into personalized interventions for PTSD management.

IP 39. *Therapeutic Potential of Histone Deacetylase Inhibitors (HDACi) in Lung Cancer: A Narrative Review of Clinical Trials*

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Lung cancer represents a global challenge in oncology. Resistance to conventional therapies underscores the need for innovation. Histone modification through acetylation is a key mechanism in the genesis of this cancer. Histone deacetylase inhibitors (HDACi) aim to restore normal gene expression and are being investigated for their synergistic potential with other treatments. Despite strong preclinical evidence, the clinical efficacy of HDACi in lung cancer has not been established. It is important to synthesize the evidence from clinical trials in order to recognize the benefit of these therapies, identify trends, and highlight research opportunities. A systematic search was conducted in PubMed and Scopus using MeSH/DeCS terms: "(HDAC Inhibitor)" AND ("Epigenetics") AND ("lung cancer"), filtered by "Clinical Trials." Of the 9 articles identified, 7 were included after exclusion based on full-text availability. Analysis of 7 trials (phases I/II) reveals: Efficacy signals, Objective responses were observed in combination with chemotherapy (Belinostat + Cisplatin/Etoposide: ORR 47% in SCLC) and isolated cases of durable responses with epigenetic therapies (Azacitidine + Entinostat: CR >1 year). The combination of HDACi + immunotherapy (Entinostat + Pembrolizumab) showed a low ORR (9.2%) but durable responses (median of 10 months). Biomarkers: Gene demethylation (APC, RASSF1a), high

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expression of E-cadherin/HDAC2, HLA-DR, and BIM deletion polymorphism. Toxicity: Hematologic and gastrointestinal toxicity emerged as the main limiting factors. HDACi act as "sensitizers" in combination therapies. Evidence suggests their use in subpopulations defined by epigenetic and immunologic biomarkers. It is essential that future research focuses on these biomarkers and on consolidating precision therapies in lung cancer.

IP 40. *Muscle doesn't forget: Epigenetic Memory Induced by HIIT*

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Skeletal muscle has the capacity to adapt to exercise through molecular mechanisms that optimize its function. Recent evidence suggests that high-intensity interval training (HIIT) can induce epigenetic memory by establishing epigenomic marks that facilitate faster readaptation during retraining. This review examined five recent studies that evaluated the effects of HIIT on the muscle epigenome. These included longitudinal human studies with muscle biopsies collected during training, detraining, and retraining phases. Analyses were performed using DNA methylation arrays, histone modification profiling, and, in some cases, integration with transcriptomic and proteomic approaches. Findings indicate that HIIT induces hypomethylation in genes regulating mitochondrial biogenesis and energy metabolism, particularly in PGC-1α, as well as in loci related to cellular signaling (ADAM19, INPP5A, MTHFD1L, CAPN2, SLC16A3). Biopsies revealed persistent PGC-1α hypomethylation, increased histone acetylation (H3K27ac) associated with greater chromatin accessibility in metabolic genes, enhanced expression of genes linked to mitochondrial biogenesis (PGC-1α) and lactate/glucose transport (SLC16A3, GLUT4), as well as evidence of cellular remodeling and increased mitochondrial proteins and oxidative enzymes. These modifications persisted for up to seven weeks of inactivity and were reactivated upon retraining, supporting the existence of muscle epigenetic memory. HIIT not only drives immediate physiological adaptations but also establishes long-lasting epigenetic programming that enhances muscle plasticity and metabolic adaptation, with potential applications in the prevention of metabolic diseases, rehabilitation, and athletic performance. Future research should aim to increase sample sizes, include more diverse populations, standardize training protocols, and extend longitudinal follow-ups.

IP 41. *Environmental Epigenetics and Metabolomics of Medicinal Plants: Resilience and Bioactive Quality in Contaminated Soils of* Puebla

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Environmental epigenetics examines how contaminants modify gene expression without altering the DNA sequence. In medicinal plants, such changes can impact the production of secondary metabolites, bioactive molecules with therapeutic properties. Metabolomics makes it possible to characterize these chemical variations and to understand how environmental factors influence plant resilience and the quality of compounds traditionally and complementarily used in medicine.

In Puebla, many rural communities rely on medicinal plants to treat common ailments. Regions such as the Mixteca and the Sierra Norte have a strong herbal tradition that has been passed down through generations. However, exposure to heavy metals and urban or industrial discharges may induce epigenetic modifications and alter the capacity of these species to produce bioactive metabolites. This situation raises concerns about the safety and effectiveness of their consumption. The present work aims to explore the interaction between environmental contamination, plant epigenetics, and the production of secondary metabolites in medicinal plants. It proposes to analyze how contaminants modify the epigenetic responses of traditionally used species and how these alterations affect the synthesis of compounds such as flavonoids, alkaloids, or terpenes. Through metabolomics, the goal is to establish a bridge between environmental changes and the bioactive quality of plant extracts, with the purpose of evaluating their implications for human health and the continuity of regional herbal knowledge. Addressing the relationship between contamination, epigenetics, and metabolomics in medicinal plants of Puebla represents an innovative line of research with both biotechnological and social relevance. This approach constitutes a novel perspective in the study of regional herbal medicine, as it integrates scientific validation with traditional knowledge, while promoting sustainable use strategies that ensure the safety and efficacy of these species and safeguard the cultural heritage of local communities.

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IP 42. *Interaction Between Gut Microbiota, Epigenetic Metabolites and the Risk of Irritable Bowel Syndrome: Current Perspectives on Its Pathophysiology and Treatment*

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Irritable Bowel Syndrome (IBS) is a multifactorial, chronic functional disorder characterized by recurrent abdominal pain and altered bowel habits. Accounting for up to 30% of gastroenterology consultations, IBS remains a major clinical challenge due to its heterogeneous presentation and diagnostic complexity. The pathophysiology of IBS involves gut microbiota dysbiosis, impaired intestinal barrier function, and immune activation. These factors, in conjunction with stress and dietary influences, may induce epigenetic modifications that contribute to disease onset and persistence. The nonspecificity of clinical manifestations hinders accurate diagnosis and increases susceptibility to clinical bias. Consequently, a deeper understanding of its molecular and epigenetic mechanisms is essential for improving diagnostic precision and therapeutic strategies. Dysbiotic shifts in IBS are characterized by reduced abundance of Ruminococcaceae and Christensenellaceae, leading to altered vitamin B6 metabolism, a cofactor critical for digestive processes and the biosynthesis of neurotransmitters such as dopamine and adrenaline. These alterations negatively impact intestinal motility, visceral hypersensitivity, and mood regulation. Furthermore, dysbiosis reduces butyrate production, a key epigenetic metabolite that inhibits histone deacetylases (HDACs), thereby enhancing the transcription of anti-inflammatory genes and maintaining intestinal barrier integrity. Emerging evidence has also revealed distinctive DNA methylation signatures that differentiate IBS from other disorders with overlapping symptoms, underscoring the interconnected roles of stress, diet, microbiota, and epigenetic regulation. Notably, low-FODMAP dietary interventions have demonstrated efficacy in modulating metabolic pathways and alleviating symptom burden. IBS represents the outcome of a complex interplay between microbial dysbiosis, metabolic disturbances, and epigenetic regulation. Targeted interventions to modulate gut microbiota and its metabolites—through nutritional strategies, probiotics, and epigenetic-based therapies—hold promise as innovative approaches for the development of personalized treatments.

IP 43. *Lead Neurotoxicity in Children: A Hidden Risk*

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Lead is a heavy metal widely distributed in the environment due to human activities, found in paints, toys, glazed ceramics, water, and batteries. Its absorption is higher during childhood and represents a serious risk for neurodevelopment, since no safe level of exposure exists. Lead exposure causes severe neurological effects in children worldwide. The lack of preventive strategies and the limited understanding of its mechanisms of damage hinder early intervention. Based on this, the following question arises: Which prevention and control measures are most effective in reducing lead-induced neurotoxicity in vulnerable populations? A qualitative, retrospective, cross-sectional, and descriptive documentary review was conducted in PubMed, Scielo, and Google Scholar, including publications in English and Spanish from the year 2000 onwards. Based on the information obtained, the sources and routes of lead entry, its neurotoxic mechanisms, and the main neurological effects in children were explained. Lead affects the brain through DNA hypomethylation, particularly of 5-methylcytosine, and mitochondrial dysfunction caused by alterations in calcium metabolism and increased oxidative stress, favoring the onset of ADHD, autism, schizophrenia, and cognitive impairment. In Mexico, 17.2% of young children present intoxication (>5 µg/dL), mainly due to leadglazed pottery, with the most affected being vulnerable, indigenous, and malnourished communities. The absence of prevention and early diagnosis aggravates the risk. It is urgent to eliminate lead in ceramics, strengthen regulation, educate the population, and search for epigenetic biomarkers for prevention.

IP 44. *Environmental Factors and Their Influence on DNA Methylation in the Development of Allergic Rhinitis*

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Allergic rhinitis (AR) is defined as inflammation of the nasal mucosa; it is an atopic disease caused by an immune response to allergens, characterized by nasal congestion, rhinorrhea, sneezing, postnasal drip, and nasal itching. It is classified as a component of the systemic allergic response, with other associated conditions sharing an underlying systemic pathology. AR results from interactions between genetic predisposition and environmental exposures. DNA methylation

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modulates the expression of immune-related genes, influences clinical severity, and reflects early-life exposures such as tobacco smoke or pollution. Changes in CpG sites of nasal epithelium and sublingual immunotherapy highlight its regulatory role. Histone deacetylation via HDAC1 enhances inflammation and weakens the epithelial barrier, although this can be reversed with inhibitors or immunotherapy. MicroRNAs fine-tune allergic responses and serve as potential biomarkers. Polymorphisms in IL-4 and IL-4R promote an exaggerated Th2 response, increasing IgE and cytokine levels, perpetuating inflammation and susceptibility. Diagnostic Implications: AR diagnosis can be approached through basic or more in-depth methods, using the following tools: Medical History, Clinical Presentation, Physical Examination, Laboratory Tests, Epigenetic Biomarkers. Environmental Factors That Modulate AR Epigenetics: 1 Prenatal exposure to pollen influences DNA methylation at birth and may be associated with the development of asthma and AR; 2 Twin and familial segregation studies show higher concordance in monozygotic twins compared to dizygotic twins, making genetic predisposition one of the strongest risk factors for developing allergic rhinitis; 3 Epigenomic studies linked to environmental exposures highlight that cigarette smoke and air pollutants modify DNA methylation patterns. Treatment: Four pillars are established for symptom management: 1 Education; 2 Allergen Avoidance, 3 Pharmacological Treatment; 4 Specific Immunotherapy

IP 45. *Brain Changes in University Students Falling in Love: a review*

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Falling in love is a response of attraction toward another person that triggers a series of physiological and neurobiological reactions. In this process, neurotransmitters are involved, and various brain regions related to reward, motivation, and emotional regulation are activated in response to visual, olfactory, or physical stimuli from the person who is the object of attraction. The aim of this review is to identify the brain changes associated with falling in love in university students. Evidence shows that, in this population group, areas such as the left anterior cingulate cortex are activated, where high peaks of regional homogeneity—indicative of greater localized activity—are observed. Likewise, an increase in functional connectivity has been documented between the insula, caudate, nucleus accumbens, and central nucleus of the amygdala. Notably, dopamine is found at high concentrations during this process, reinforcing the role of reward circuits in the experience of love. In conclusion, the brain plays a fundamental role in falling in love, which should not be understood solely as an emotional experience but also as a complex neurobiological process that integrates physiological, cognitive, and affective aspects.

IP 46. *Impact of Hyperandrogenism on Fetal Neurodevelopment During Pregnancy in Women with Polycystic Ovary Syndrome*

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According to WHO data, in 2025 polycystic ovary syndrome (PCOS) will affect between 6% and 13% of women of childbearing age, and up to 70% are undiagnosed. One of the main symptoms is hyperandrogenism with elevated levels of certain hormones, which can cause genetic alterations at the neuronal level in the fetus of pregnant patients with PCOS. To compile available scientific evidence on how maternal hyperandrogenism associated with PCOS can cause genetic alterations in the fetus's neurons. A systematic search was conducted using the following keywords: "Neuronal gene expression," "Fetal epigenetics," and "Polycystic ovary syndrome (PCOS)" on academic platforms and dissemination sites, obtaining a total of 62 articles, which were subjected to temporary language and thematic inclusion and exclusion criteria, resulting in a total of 25 articles that were read and analyzed. Fetal hyperandrogenism induces reprogramming of the hypothalamic-pituitary-gonadal axis and neuroendocrine function through epigenetic mechanisms, mainly through modifications in DNA methylation. These changes result in alterations in the expression of key genes: an increase in TIAL1 and FABP5, and a reduction in RNF141 and INIP. At the neuronal level, it particularly affects KNDy and GABAergic neurons, modulating their activity and gene expression. Together, they directly impact the neuroendocrine programming of the fetus, with repercussions on reproductive, metabolic, and long-term hormonal regulation processes. Maternal hyperandrogenism characteristic of patients with PCOS affects fetal neuronal development at the genetic level, mainly through epigenetic alterations such as DNA methylation. It therefore creates an adverse intrauterine environment that alters the genetic and epigenetic programming of fetal neuronal development, with lasting functional and phenotypic consequences.

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IP 47. Epigenética y determinantes sociales de la salud: contaminación y exposición a tóxicos.

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The increasing exposure to environmental pollutants like pesticides, microplastics, and bisphenol A (BPA) has led to a re-evaluation of their impact on human health, showing they act as epigenetic disruptors. This review synthesizes evidence that these underlying molecular changes, which don't alter the DNA sequence but do affect its function, are linked to the development of diseases. The poster addresses how contaminants influence epigenetic mechanisms like DNA methylation and histone modifications, connecting exposure to specific pathologies, the potential for transmission to future generations, and the capacity for epigenetic adaptation. To review recent scientific evidence on exposure and epigenetic changes. A qualitative literature review was conducted on 8 articles, selected from 20 reviewed, focusing on epigenetics as a determinant of health, specifically in its relationship with pollution and exposure to toxins. The search was performed in scientific databases like PubMed, Scopus, and SciELO. Evidence shows that contaminants act as epigenetic disruptors, modifying gene expression. These changes are associated with respiratory, allergic, and neurodegenerative diseases and can be transmitted transgenerationally. In addition, some studies describe possible epigenetic adaptations to chronic exposure, which may be protective or, conversely, increase susceptibility to diseases.

IP 48. *Impact of Stress on Academic Performance in University Students: A Review*

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Stress is a physiological process primarily characterized by the release of glucocorticoids in response to different stressors. In the university setting, students face multiple transitions and demands—such as migration to new cities, separation from their family environment, and academic or practical overload—that can trigger acute, episodic, or chronic stress responses. Chronic stress is particularly relevant, as it decreases neuroplasticity by damaging neurons and synaptic connections, affecting neurogenesis in the hippocampus. This process is mediated by sustained activation of the hypothalamic-pituitary-adrenal (HPA) axis, which leads to the release of hormones such as corticotropin-releasing hormone (CRH), adrenocorticotropin hormone (ACTH), and cortisol. At the molecular level, prolonged exposure to cortisol alters gene expression through epigenetic mechanisms, including DNA and histone modifications, which increases vulnerability to disorders such as anxiety, depression, and poor academic performance. Several studies suggest that interventions such as moderate physical activity and cognitive behavioral therapy can positively modulate the HPA axis and promote the epigenetic regulation of genes related to neuronal plasticity and emotional control. These findings highlight the importance of understanding the impact of stress on university students not only from the perspective of mental health and academic performance but also in its epigenetic dimension, opening new opportunities for the development of preventive and therapeutic strategies.

IP 49. *Artificial Intelligence in Breast Cancer Screening: Diagnostic Advances and Implementation Challenges.*

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Breast cancer is the most common malignancy in women and a major cause of mortality, making its early detection a public health priority. In Mexico, more than 23,000 new cases are reported annually, underscoring the urgent need for innovative diagnostic strategies. Artificial intelligence (AI), particularly through deep learning algorithms, has shown great potential to enhance diagnostic accuracy and optimize screening programs. The objective of this review was to analyze recent evidence on AI applications in breast cancer detection using medical imaging. International databases (Web of Science, Elsevier, Access Medicine) were consulted, selecting articles published between 2020 and 2025 that evaluated AI performance in mammography, tomosynthesis, ultrasound, and magnetic resonance imaging. Findings indicate that convolutional neural network–based models achieve performance comparable or superior to expert radiologists, reducing false negatives and positives while improving sensitivity in dense breasts. Furthermore, Computer-Aided Detection (CAD) systems integrated with AI have demonstrated the ability to reduce workload without compromising diagnostic quality. Nevertheless, challenges remain, including limited external validation, lack of algorithm interpretability, absence of standardization, and regulatory barriers, which hinder clinical implementation. In conclusion, AI represents a promising and complementary tool for early breast cancer detection. Safe and equitable adoption requires prospective studies, the development of more transparent models, and strategies to facilitate integration into healthcare systems with limited resources.

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IP 50. *Motivation as a Determining Factor in University Students' Learning*

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Motivation is a psychological and neurobiological process that drives human decision-making and behavior throughout life. In the educational domain, it plays a decisive role in knowledge acquisition and competence development. Motivation is commonly classified into two main dimensions: intrinsic motivation, related to personal interest and internal satisfaction, and extrinsic motivation, associated with external factors such as rewards, grades, or social recognition. Both dimensions constantly interact in the university context, influencing career choice, academic persistence, and performance. From a physiological and neurobiological perspective, motivation involves the activation of brain circuits linked to the reward system, particularly in regions such as the nucleus accumbens, prefrontal cortex, and hippocampus. Neurotransmitters including dopamine, serotonin, and norepinephrine regulate pleasure, attention, and memory-processes directly involved in learning. Dopamine, in particular, reinforces goal-directed behavior and strengthens positive feedback, while the hypothalamic-pituitary-adrenal (HPA) axis modulates stress responses that impact motivation and students' ability to focus. Within this framework, playful pedagogy emerges as a teaching-learning strategy that stimulates motivational circuits through dynamic and engaging experiences. The challenge lies in designing activities that are sufficiently enjoyable to sustain attention and commitment, without neglecting the ultimate goal: fostering meaningful learning. This review aims to explore the psychological, social, and neurobiological factors that increase or diminish university students' motivation and their impact on academic engagement. Understanding how cognition, emotion, and physiology.

IP 51. *The Biological Clock Under the Epigenetic Magnifying Glass: Aging Mechanisms and Their Induction by Contaminants*

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Chronological age generally refers to the actual years a person lives, and biological age to the physiological state and function of an individual. Interest in investigating the epigenetic mechanisms of aging and the development of epigenetic clocks has been growing due to their usefulness as public health markers. Systematic comparison of bibliographic works focused on biomarkers of aging, as well as the impact that pollution has on adulthood. Bibliographical review in the PubMed database from 2015 to 2025, using keywords such as "epigenetics, aging, biological clock, geriatric diseases, and environmental pollution" for a more relevant search. Epigenetic clocks are mathematical algorithms that predict a subject's chronological age by measuring the levels of methylated DNA (mDNA) at multiple sites of cytosine-phospho-guanine (CpG) dinucleotides in their genome. The genetic clock estimates real age by measuring DNA methylation and is more effective than transcriptomic and proteomic data or telomere length. Changes in methylation patterns impact gene expression and other cellular processes, developing several age-related human pathologies: neurodegenerative, cancer, cardiovascular, metabolic, and osteoarthritis. Exposure to air pollutants can accelerate this process through the generation of reactive oxygen species, inflammation, alteration of the cutaneous microbiome, and activation of the aryl hydrocarbon receptor. Solar radiation also has an impact on the skin, producing a synergistic effect with air pollution («photocontamination»). So far the most cited epigenetic clocks are Horvath, Hannum, GrimAge, PhenoAge, DunedinPACE, and micronucleus formation as a genotoxic biomarker micronucleus formation. More potent and common determinants of biological age need to be identified to develop and validate interventions that can slow down or counteract the aging process and associated pathologies. A potential strategy to influence aging is to intervene in lifestyle (diet and physical activity) and nutrition, representing one of the most promising approaches to extend the period of health and achieve longevity. Biological clocks allow us to investigate geriatric pathologies through epigenetic studies, making it possible to analyze environmental factors that trigger them and look for alternatives to delay this process. As well, identify the "epigenomic identity card" of each disease and even of each patient.

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IP 52. *Updates on Neurofibromatosis Type 1, Literature Review*

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Neurofibromatosis type 1 (NF1) is an autosomal dominant, multisystemic genetic disorder, considered the most common cancer predisposition syndrome. It originates from inactivating mutations in the NF1 gene, located at 17q11.2, which encodes neurofibromin. This protein negatively regulates RAS; its loss leads to constitutive activation of the RAS/MAPK pathway, promoting uncontrolled cell proliferation and tumor formation. NF1 presents a clinical challenge due to its variable phenotypic expression, which includes café-au-lait spots, cutaneous neurofibromas, bone complications, and central nervous system tumors. Approximately half of the cases arise from de novo mutations, which complicates early diagnosis. Traditional management has been based on clinical surveillance and symptomatic treatment, with limited options for controlling tumor progression, especially in plexiform neurofibromas with a risk of transformation into malignant peripheral nerve sheath tumors (MPNSTs). The diagnosis is based on internationally validated clinical diagnostic criteria. In recent years, the therapeutic approach has changed with the introduction of targeted therapies. MEK inhibitors, such as selumetinib, have demonstrated efficacy in reducing the volume of inoperable plexiform neurofibromas, representing the first specific medical alternative. Likewise, current studies highlight the influence of epigenetic mechanisms and metabolic alterations on the clinical expression of the disease. NF1 is a complex disease that requires lifelong monitoring and multidisciplinary care. Understanding its pathophysiology has driven innovative therapies beyond symptomatic control. The integration of RAS/MAPK pathway inhibitors represents a significant advance in improving quality of life. Future research aims to combine molecular inhibitors and metabolic modulators, as well as explore epigenetic therapies. Strengthening specialized centers for genetic diagnosis, clinical trials, and equitable access to treatments is essential to providing comprehensive care and ensuring better outcomes for affected patients.

IP 53. *Identification of Genes Related to the Prevention of Adverse Drug Reactions*

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Adverse drug reactions (ADRs) are unjustified and unexpected responses to medications, which can range from mild to severe and occasionally be fatal. Their global incidence is approximately 8%. ADRs increase morbidity, prolong hospitalization, and raise healthcare system costs. In Puebla, the prevalence of adverse drug events was reported at 3.6%, highlighting that the medications most frequently involved were antibiotics and anti-inflammatory drugs. Proof of their great impact on the population is the PROY-NOM-220-SSA1-2024, in which, through the National Pharmacovigilance Center, investment must be made in acquiring the necessary tools for implementing this adjustment in the reporting process. The "Yellow Card" system identified the genes CYP2C19, CYP2D6, and SLCO1B1 associated with the prevention of ADRs. This was achieved by using cross-referenced lists of drugs against ADR events, finding that only 12 genes were involved in preventable adverse reactions, and 3 of them were responsible for 75% of such preventable reactions. The prevalence of prescriptions was compared with ADR reports, adjusting for pathological condition, gender, and associated gene. It was found that 47% of patients were under psychiatric treatment, while 24% were receiving medication for cardiovascular diseases. Regarding gender, the most affected patients were males, presenting severe but non-fatal ADRs. Meanwhile, 75% were related to three genetic variants CYP2C19, CYP2D6, and SLCO1B1, which modify enzymes and proteins of metabolism and therefore affect drug transport. The clinical correlation of ADRs with these genes represents progress in their prevention. Therefore, the standardization of these tests in the future will benefit a larger population.

IP 54. *Use of Proteomics in the Identification of Biomarkers in Neurodegenerative Diseases*

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Neurodegenerative diseases are characterized by the progressive loss of neurons and cognitive or motor functions. They are defined as chronic and incurable disorders that generate progressive disability. Globally, they affect more than 50 million people, with an expected increase driven by population aging. These pathologies generate a major social impact by causing dependency and the need for long-term care. Medically, current treatments are only symptomatic. This increases

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direct costs related to hospitalization and pharmacological therapies, as well as indirect costs due to work loss and family burden. In Mexico and Latin America, the situation is critical because of limited health system resources. Furthermore, incidence is rising in association with increased life expectancy. This scenario highlights the urgent need for novel diagnostic and therapeutic approaches. Proteomics has enabled the identification of plasma and cerebrospinal fluid biomarkers such as phosphorylated tau, neurofilament light chain (NfL), and glial fibrillary acidic protein (GFAP), which detect early changes before clinical symptoms in Alzheimer's and Parkinson's disease. In carriers of autosomal dominant mutations, proteomic trajectories have been mapped, reflecting disease progression from beta-amyloid accumulation to synaptic loss and neuroinflammation. These profiles have been integrated with PET neuroimaging, strengthening the correlation between molecular changes and clinical progression. Proteomics has also revealed the relevance of inflammation and glial dysfunction as key drivers of neurodegeneration. Recent advances in single-cell and spatial proteomics provide greater resolution of these processes, allowing the identification of vulnerable cellular populations and altered pathways. Proteomics emerges as a fundamental tool for early diagnosis and patient stratification. In the coming years, the integration of proteomics, neuroimaging, and artificial intelligence may pave the way for the proposal of new therapeutic targets.

IP 55. *Beckwith-Wiedemann Syndrome as a Consequence of Epigenetic Alterations in Assisted Reproduction*

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Beckwith-Wiedemann syndrome (BWSp) is an imprinting disorder characterized by overgrowth, congenital malformations, and tumor predisposition. Its most frequent molecular basis involves epigenetic alterations in the 11p15.5 region, which contains the ICR1 and ICR2 imprinting control centers. Most patients exhibit loss of methylation in ICR2 (IC2-LOM) or gain in ICR1 (IC1-GOM), mosaicism, or paternal uniparental disomy (pUPD). Studies have shown that children conceived through assisted reproductive technology (ART) have an increased risk of developing BWSp, with a marked predominance of IC2-LOM as the molecular alteration. Comparisons between naturally conceived and ART-conceived cohorts confirm that the distribution of subtypes differs, with a lower frequency of IC1-GOM and a higher proportion of IC2-LOM in ART. The most widely accepted hypothesis is that manipulation of gametes and embryos during ART interferes with the establishment and maintenance of epigenetic marks in the preimplantation stage. Furthermore, multilocus imprinting (MLID) alterations have been described in a proportion of cases, suggesting a more global vulnerability of the epigenome. International cohort studies have confirmed phenotypic variability according to the type of alteration: cases with IC2-LOM tend to have a milder phenotype, while IC1-GOM and pUPD are associated with a higher tumor risk. The identification of microdeletions in IC1 provides evidence of rare familial mechanisms. Likewise, reports of tissue mosaicism reveal that epigenetic burden can modulate clinical expression. Taken together, the accumulated evidence reinforces the connection between ART and BWSp, primarily through alterations in ICR2. The finding of MLID in some cases, along with the involvement of maternal genes and the observation of microdeletions in ICR1, suggests that ART may act as a trigger on a predisposed genetic or epigenetic terrain. Recognizing these mechanisms is fundamental for genetic counseling, clinical surveillance, and the development of safer ART protocols.

IP 56. *Use of Infrared Thermography in Necrotizing Enterocolitis in Patients in the Neonatal Intensive Care Unit*

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Necrotizing enterocolitis is a serious neonatal condition characterized by intestinal ischemic necrosis, severe inflammation, and bacterial proliferation with gas formation and dissection. Mortality is inversely related to gestational age and birth weight, reaching up to 100% in advanced stages. Survivors frequently present with permanent sequelae, especially after surgical management, including growth retardation, neurodevelopmental delay, and gastrointestinal disorders. Diagnosis is based on clinical manifestations—abdominal distension, bile vomiting, and rectal bleeding—complemented by radiographic findings and the Bell classification as the diagnostic standard. Thermography emerges as a noninvasive, radiation-free alternative for the diagnosis and monitoring of necrotizing enterocolitis by detecting early variations in abdominal temperature. Its implementation could reduce risks in vulnerable neonates and optimize monitoring in intensive care, enhancing diagnostic accuracy compared to conventional methods. This work analyzes the usefulness of infrared thermography for diagnosing necrotizing enterocolitis during the early stages of the disease. It presents the physical and mathematical foundations for standardizing the use of the infrared camera. These principles can be applied in any Neonatal Intensive Care Unit in our country.

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IP 57. *When Trauma Leaves a Mark on DNA: PTSD and Biological Aging*

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Post-traumatic stress disorder (PTSD) is a psychiatric condition that arises after experiencing traumatic events. It is characterized by core symptoms such as hypervigilance, avoidance, and the re-experiencing of trauma. Recent evidence shows that PTSD not only affects individuals' mental health but also leaves biological marks, particularly on telomere length, which serves as an indicator of accelerated biological aging. Ratanatharathorn et al. (2023) found that individuals with PTSD have shorter leukocyte telomeres—on average, 270 base pairs shorter—equivalent to approximately 7.6 years of additional cellular aging, even after adjusting for depression and lifestyle factors. This finding suggests that traumatic stress has a direct effect on a person's biological age. Carvalho et al. (2022) observed that in women who had experienced sexual assault, early-onset re-experiencing symptoms of PTSD were associated with telomeres that were 1.1 kb shorter, demonstrating that the cellular effects of trauma can appear rapidly. Zhou et al. (2023) reported that childhood maltreatment was associated with telomere shortening of more than 0.15 kb, regardless of sociodemographic or mental health factors. This indicates that the impact of trauma can persist throughout the lifespan. Rajkumar (2025) concluded that these telomeric changes are driven by mechanisms related to chronic stress, persistent inflammation, and mitochondrial dysfunction, and may have transgenerational consequences. The synthesis of the evidence suggests that PTSD may accelerate biological aging through the mechanism of telomere shortening. These findings have important clinical implications, indicating that treating PTSD could be an intervention that not only improves mental health but also mitigates biological damage and reduces the risk of transmission across generations.

IP 58. *Use of Multiomics and Artificial Intelligence as Support for Decision-Making in the Diagnosis of Insulin Resistance*

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Childhood obesity is on the rise, with an estimated 400 million children living with obesity by 2035. Insulin resistance (IR) is a metabolic comorbidity that increases the risk of type 2 diabetes and cardiovascular disease. Childhood obesity and insulin resistance are a growing public health problem, with effects on quality of life and healthcare costs. This increases the risk of metabolic complications in adolescents and adults. Traditional biomarkers have limited predictive capacity, making early detection of at-risk children difficult. Therefore, it is essential to develop more accurate diagnostics. Emerging technologies, such as explainable artificial intelligence (XAI) and multi-omic data, together offer opportunities to improve the identification and prevention of these conditions. This study proposed the follow-up of pubertal pediatric patients for three years. Anthropometric data were collected. Biomarkers of insulin resistance, cardiovascular/inflammatory proteins, and adipokines were obtained from blood samples. In addition, a panel of haplotypes was performed, which would be analyzed by AI software using the Minimic 4 algorithm through the cloud-based interface of the Michigan Imputation Server. Thus, the combination of genomic (Gen), epigenomic (Epi), and clinical (Clin) data analysis of pediatric adolescents was able to more efficiently predict risk data for obesity and insulin resistance. The combination of clinical and epigenetic data offers high predictive power for identifying IR in children with obesity. Therefore, artificial intelligence will improve medical confidence and discover novel biomarkers.

IP 59. *The Impact of Sleep on Memory Consolidation: Insights from Educational Neuroscience and Epigenetics*

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The relationship between sleep and memory is a central topic in educational neuroscience, as both processes directly influence learning. Sleep regulation depends on the interaction of circadian rhythms, stress levels, and neuronal plasticity. During slow-wave and REM sleep, memories are consolidated through the reactivation of hippocampal and neocortical circuits, in which neurotransmitters such as acetylcholine, dopamine, and serotonin play a crucial role. Synaptic plasticity in these regions is also modulated through epigenetic mechanisms, including histone acetylation and DNA methylation,

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which influence the encoding, consolidation, and retrieval of information. In university students, academic demands, complementary work activities, psychoactive substance use, and intensive use of digital technologies increase the prevalence of sleep disturbances, directly impairing memory and academic performance. Learning involves the integration of perception, attention, memory, comprehension, and reasoning, as well as the transfer of information from working memory to long-term memory, a process vulnerable to sleep deprivation or poor sleep quality. A systematic review was conducted in PubMed and SciELO following PRISMA guidelines, focusing on observational studies published between 2019 and 2025. Twenty journal articles were selected that examined variables related to sleep quality, daytime sleepiness, and sleep disorders. Evidence indicates that insufficient or poor-quality sleep produces excessive daytime sleepiness and reduces synaptic plasticity, leading to deficits in attention, memory consolidation, and executive functions, which significantly impact academic performance. In conclusion, sleep problems represent a critical factor in higher education, with neurobiological and epigenetic implications that condition students' memory and learning capacity.

IP 60. *Low Oxygen Levels in Tumors Could Enhance Some Immune Responses Against Cancer*

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Cancer is a disease characterized by the uncontrolled growth of cells that generate a tumor microenvironment. According to the WHO, in the next two decades the number of cancer cases could increase by 60% to 80% in low- and middle-income countries. Hypoxia has traditionally been considered a factor that drives cancer's aggressiveness and resistance to treatment. This is because it not only forces tumor cells to adapt and survive, but also induces immune cells to tolerate and promote tumor growth. As a result, patients often face a worse prognosis. At a scientific level, this implies the need to rethink established knowledge and open new lines of research. Recent research has identified a population of hypoxic macrophages that are more effective against cancer. These macrophages present epigenetic alterations and the participation of factors such as NF- ΔB and HIF1Δ, which activate genes related to inflammation. In cases of bladder and ovarian cancer, those containing hypoxic macrophages showed better clinical outcomes. This means that, although hypoxia promotes tumor growth in most cases, it can also generate conditions in which the immune system responds more strongly. Therefore, this finding expands the understanding of the relationship between the tumor microenvironment and the body's defenses. Finally, this discovery opens the possibility of designing innovative therapeutic strategies that take advantage of the plasticity of macrophages and inflammatory responses for the benefit of patients. The identification of hypoxic macrophages may serve as a basis for developing immunotherapies that leverage hypoxic conditions to treat cancer.

IP 61. *Fructose Intake During Pregnancy Could Enhance the Negative Effects of the Western Diet on Offspring*

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Fructose is a carbohydrate present in large quantities in processed foods and sugary drinks. Excessive consumption has been associated with the development of type 2 diabetes, insulin resistance, and cardiovascular disease. The incidence of these metabolic diseases has increased significantly, with more than 60% of overweight or obese adults in industrialized countries. Excessive fructose consumption has been linked to an increase in metabolic diseases. Studies have shown that maternal nutrition can program the metabolism of offspring. Fetal programming induced by fructose and a Western diet could have long-term consequences for metabolic health. Therefore, our objective is to present evidence based on a mouse model of high-calorie diet consumption during pregnancy. Elena Fauste et al., in 2024, studied the offspring of rats fed 10% fructose-containing water (MF) during pregnancy and a control group (MC). At three months, some offspring received a Western diet (2% cholesterol + liquid fructose) for 21 days. The MF offspring consumed less fructose and cholesterol than the MC offspring, but eliminated more cholesterol in bile. However, they had greater intestinal lipid absorption, associated with increased expression of transporters such as CD36 and SR-B1. They also showed hypersensitivity to the GLP2 peptide, which promotes fat absorption and chylomicron formation. These findings indicate that fetal programming alters intestinal function, increasing metabolic risk. Future case-control studies in humans would help explain why some individuals who eat unhealthy diets in smaller quantities and sporadically are more likely to develop intestinal and metabolic diseases. More stringent dietary recommendations for pregnant women are needed.

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IP 62. *Epigenetic Alterations and Structural Brain Changes in Schizophrenia: A Systematic Review*

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Schizophrenia is a complex psychiatric disorder with positive, negative, and cognitive symptoms. Its etiology is associated with neurodevelopmental processes where epigenetics (particularly DNA methylation, histone modifications, and microRNAs) plays a key role in gene regulation without altering the DNA sequence. Despite advances in neuroimaging, a gap remains in understanding how epigenetic mechanisms impact the brain structure of patients with schizophrenia. Identifying this relationship is fundamental to explaining clinical variability and proposing future therapeutic targets. A systematic review was conducted in PubMed, Scopus, and Web of Science (2020–2025). Original studies in humans that analyzed epigenetic mechanisms associated with brain structures using structural magnetic resonance imaging, voxel-based morphometry, and diffusion tensor imaging were included. Reviews and animal studies were excluded. The findings highlight DNA methylation as the most frequently reported mechanism, primarily in the RELN, BDNF, DRD1, and XPO1 genes, which are associated with hippocampal volume reductions. The prefrontal cortex showed cortical thinning related to C4A overexpression, while microRNAs (miR-134, miR-138, and miR-223) were linked to alterations in the hippocampus, amygdala, and cerebellum. These results support a neurodevelopmental model in which epigenetic mechanisms modulate the maturation of limbic-prefrontal circuits and white matter. The evidence suggests that DNA methylation and microRNAs are key mediators of structural alterations in schizophrenia. However, methodological heterogeneity limits the generalizability of these findings. Longitudinal and multimodal studies are needed to clarify the causal relationship between epigenetics, neuroanatomy, and clinical manifestations.

IP 63. Importance of Genomic Diagnosis in Reproductive Medicine and Assisted Reproduction

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Reproductive genetics studies the hereditary mechanisms that influence fertility, gametogenesis, embryonic development, and reproductive capacity. Approximately 15% of couples of reproductive age experience infertility problems related to genetic factors. Genetic infertility is caused by aneuploidies, mutations, and structural chromosomal abnormalities. This represents a medical and social challenge, as it directly affects quality of life and family dynamics. Medically, diagnosing genetic causes is vital for establishing appropriate treatment and providing genetic counseling to the patient. In recent years, advances have been made in reproductive genetic diagnosis with karyotype and FISH techniques, which have helped to identify chromosomal abnormalities in infertile patients. In vitro fertilization techniques, complemented by molecular biology, have made it possible to detect point mutations in genes related to spermatogenesis or oogenesis. Thanks to all this, the genetic diagnosis of pre-implantation embryos has become consolidated as a fundamental tool in assisted reproduction. This allows the selection of embryos free of chromosomal abnormalities or monogenic mutations before implantation. Together with next-generation sequencing, it has expanded the possibilities of detecting variants related to male and female infertility. These advances have improved the success rates of in vitro fertilization procedures. Reproductive genetics will play a key role in understanding the causes of infertility and improving diagnostic and treatment strategies. In the future, the integration of genomic sequencing and gene editing could revolutionize reproductive medicine, providing more personalized and safer solutions.

IP 64. *The Environmental Case of the Atoyac River, in* Puebla, México

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The Atoyac River, located in Puebla, Mexico, has historically been a source of agricultural, economic, and social development; nevertheless, it is currently facing a severe environmental and public health crisis due to its alarming level of contamination. This river, which was once regarded as a vital and valuable natural resource, has now turned into a significant risk factor for both human well-being and the surrounding ecosystems, since it constantly receives untreated industrial, agricultural, and domestic discharges. Such effluents contain heavy metals, toxic chemicals, and

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large amounts of organic matter that disrupt the natural balance of the river and directly affect the health, safety, and livelihoods of nearby communities. The main causes behind this critical situation include uncontrolled and accelerated industrial growth, the absence of properly functioning wastewater treatment plants, the excessive use of fertilizers and pesticides in farming, as well as the poor management of urban sewage systems. Scientific and environmental studies have demonstrated that the Atoyac River contains concentrations of lead, arsenic, and mercury that far exceed national standards and international recommendations, which are directly linked to the increase of gastrointestinal, respiratory, and dermatological diseases, in addition to severe conditions such as kidney failure, leukemia, congenital malformations, and different types of cancer. This reality highlights not only a dramatic ecological deterioration but also a serious public health emergency that threatens the quality of life of thousands of people. The origins of this degradation are associated with weak governmental control, corporate irresponsibility, and the lack of genuine social awareness. Therefore, it is urgent to implement macro-level measures, such as the strict enforcement of environmental regulations, the restoration and modernization of sanitation processes, and the continuous monitoring of water quality; together with micro-level actions that promote environmental education, citizen awareness, and strong community participation. Supported by documentary research and bibliographic analysis, these strategies seek to rescue an essential ecosystem, protect human health, and recover the balance between community and nature.

IP 65. *Advantages of Exome Analysis in Adult Patients with Rare Diseases*

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Clinical genetics is a branch of medicine that has traditionally focused on diagnosing rare diseases. Currently, exome sequencing — the process of sequencing the protein-coding regions (exons) of a patient's genome — is used to search for mutations associated with genetic and rare diseases. The paradigm that genetic and rare diseases only affect children must change. Often, the complications that lead to adults being admitted to the ICU are genetic in origin, even though they are diagnosed as idiopathic. Therefore, clinical guidelines and greater access to genomic medicine are needed to detect these cases, which are estimated to account for up to 5% of ICU admissions. A lack of access to standardized genomic medicine leads to disparities in care. This paper therefore seeks to present evidence on the advantages of exome analysis for genetic diagnosis. This retrospective study analyzed the entire exome of 365 ICU patients aged 18–40, finding that 24.4% had a genetic variant that explained their condition. The highest diagnostic performance was observed in cases of pulmonary disease (81.8%), followed by vascular disease (39.1%) and renal disease (36.8%). The most frequently implicated genes were associated with cardiac (18.9%), vascular (16.8%), and cancer (16.8%) phenotypes. The study also revealed an imbalance in the documentation of genetic diagnoses between white patients (63.1%) and black patients (22.7%). Whole exome sequencing in young ICU patients has a high diagnostic success rate, justifying its integration as an essential tool in the care of critically ill patients.

IP 66. *Use of CRISPR-Cas9 in Modulation of Inteleukin 1 Expression for Therapeutic Use*

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The function of immune responses is regulated by gene methylation. Interestingly, genetic research has developed new technologies such as CRISPR-Cas9, which has enabled the modification of genes. Altered immune responses are a significant problem in the development of cancer, as well as autoimmune diseases, where the expression of Interleukins (ILs) participates in the development of these pathologies. The use of CRISPR-Cas9 technology has allowed for the modification of gene expression, and this presents an opportunity to understand and work on cellular responses. The objective of this work is to provide evidence of gene expression modification using CRISPR-Cas9. The modification of the specific response of IL-1 was studied using the CRISPR tool in a myeloid lineage derived from B cells. It was found that the methylation of the IL-1 promoter regions could modify the expression of responses depending on the methylation of these areas. Consequently, it has been shown that it was possible not only to increase their expression by demethylation, but also to decrease it by hypermethylation. The hypermethylation of these genes not only induced the cell's inability to differentiate into the myeloid lineage but also increased its phagocytosis capacity and its activity in general inflammatory responses. Although this technique represents a therapeutic window in vitro, the complexity of the techniques and the absolute control required for experimentation in cellular and animal models still pose challenges. However, genetic modification will be a true bridge and a less ambiguous area on the path toward future therapies.

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IP 67. *Breastfeeding as a Modulator of Epigenetic Methylation of Metabolic Genes FTO and LEP in Early Childhood*

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Exclusive breastfeeding during the first six months is associated with long-term metabolic and developmental benefits. Early nutrition influences growth programming and adipogenesis through epigenetic mechanisms. FTO and LEP genes regulate energy metabolism and fat accumulation, and their methylation patterns have been linked to increased risk of childhood obesity. In Mexico, only 33% of infants receive exclusive breastfeeding, while childhood obesity prevalence is high. Despite its epigenetic impact on metabolic genes, few studies link breastfeeding with FTO and LEP regulation in early childhood, limiting preventive strategies. Breastfeeding modulates the epigenetic programming of LEP and FTO, affecting early obesity. In LEP, breastfed infants show promoter hypermethylation, reducing expression and preventing hyperleptinemia and leptin resistance [6]. Exosomal microRNAs such as miR-26a regulate adipocyte differentiation, while miR-148a and miR-21 modulate epigenetic enzymes (DNMT1, TET2). In FTO, exclusive breastfeeding mitigates the effect of the risk allele rs9939609-A through epigenetic changes in intronic regions interacting with IRX3/IRX5. The first 1000 days are a critical window for interventions that reprogram growth and metabolism via epigenetic mechanisms. Exclusive breastfeeding acts as an epigenetic modulator of LEP and FTO, reducing obesity risk through promoter hypermethylation and exosomal microRNAs. Effects are particularly relevant in carriers of risk alleles, modulating growth and adiposity. Breastfeeding is therefore a key early-life strategy for obesity prevention and healthy metabolic programming.

IP 68. *Micronuclei: Small Structures, Major Indicators of DNA Damage*

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Failures in the response to damaged DNA or chromosomal imbalances during mitosis can lead to genomic instability, causing the formation of abnormal extranuclear structures known as micronuclei (MN). MNs are masses of chromatin formed by errors during chromosome segregation, from fragments of chromatids or complete chromosomes that fail to incorporate into the main nucleus during cell division. These bodies, although similar to the nucleus in texture, staining, and focal plane, are smaller, independent, and surrounded by an imperfect membrane. Their presence reflects a high risk of genomic rearrangements that can affect various cellular functions, triggering apoptosis, senescence, cell cycle arrest, repair system failures, accumulation of cytoplasmic damage, and overproduction of free radicals. In addition, they can activate inflammatory processes and chronically maintain the innate immune response. Therefore, the evaluation of micronucleated cells has become a preventive biomarker in the early detection of genetic damage, and is especially useful in populations exposed to genotoxins and environmental pollutants. To discuss the implementation of the micronucleus technique as a non-invasive test for the early detection of DNA damage, with population application in cases of prolonged exposure to environmental pollutants. Epidemiological studies have shown that environmental pollution has a direct correlation with health problems, having a significant social impact. Epigenetic damage is one of the most promising biological mechanisms in carcinogenesis due to air pollution. In this review, we propose using MN as a sensitive, non-invasive, economical, and effective tool as a biological monitor of populations in the state of Puebla that are constantly exposed to environmental pollutants. Genetic damage is monitored using the oral mucosa of exposed individuals, detecting not only the presence of micronuclei but also other nuclear abnormalities. Micronuclei are a benchmark used as a biomarker to detect genetic damage and thus prevent disease in high-risk populations and avoid chronic health effects.

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IP 69. *Interaction Between Epigenetics and Smoking in Lung Carcinogenesis: a Review of the Evidence*

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Cancer is a complex disease that occurs when a normal cell acquires the ability to divide progressively, rapidly, and uncontrollably, losing the capacity for apoptosis. Lung cancer (LC) is the leading cause of cancer deaths worldwide, accounting for 30% of all cancer deaths. Its survival rate ranges from 10-20% due to late diagnosis. Smoking has been identified as the primary cause of LC since 1950, responsible for more than 80% of cases. The term epigenetics refers to mechanisms that alter gene expression without modifying the underlying DNA sequences, which are crucial for biological processes such as cell development and differentiation. Chronic exposure to various forms of stress can cause epigenetic alterations and lead to the development of cancer. Cigarette smoke contains more than 60 carcinogens, including tobacco-specific nitrosamines. The objective of this research is to review the epigenetic-smoking interaction in lung carcinogenesis, in order to understand the molecular mechanisms induced by chronic exposure to cigarette smoke. From tobacco. These tumors are characterized by epigenetic alterations: (a) DNA hypermethylation: observed in CpG islands within transcription start sites in the promoter regions of tumor suppressor genes. Some examples include RASSF1A, CDKN2A/p16, DAPK, and DH13. (b) Altered histone acetylation: MALAT1 and HOTAIR; key in the growth, proliferation, and metastasis of malignant cells. (c) Aberrant microRNA expression. The interaction between epigenetics and smoking is fundamental in lung carcinogenesis. Epigenetic changes are early and crucial events induced by smoking, offering promising avenues for new lung cancer biomarkers and therapeutic strategies. The interaction between epigenetics and smoking is fundamental in lung carcinogenesis; it is highlighted that epigenetic changes are early and crucial events induced by smoking; this offers promising avenues for new lung cancer biomarkers and therapeutic strategies.

Las revistas médicas se han integrado a un sistema internacional, iniciado en 1978 en Vancouver, Canadá que prosiguió después de 1997 por el International Committe of Medical Journal Editors, (ICMJE), con el objeto de uniformar el estilo de redacción de los artículos biomédicos, para mejorar su claridad.

El objeto de estas instrucciones es con claridad y facilitar su publicación en la **Revista de la Sociedad Mexicana de Toxicología**, que acepta publicar, Editoriales, artículos originales, artículos de revisión, casos clínicos, artículos especiales, cartas al Editor.

Todos los trabajos deben dirigirse al Editor: revistasomtox@somtox.com.mx

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1. **Página inicial:**
   a. Título en español con mayúsculas
   b. Título en inglés con mayúsculas
   c. Todos los autores con nombre completo. Si hay más de 10 autores, terminar con "y cols" (*et al* en artículos en inglés)
   d. Señalar grado académico y sitio de trabajo de los autores; direcciones completas con población, teléfonos y e-mail del primer autor.
   e. Titulo corto de 10 espacios o 3 palabras.
2. **Texto completo elaborado en archivo de word**, tamaño carta, espacio de renglón de 1.5, letra Arial tamaño 12. El trabajo debe tener máximo 8 páginas de texto sin contar referencias. Los Editoriales son por invitación
3. **Referencias bibliográficas** recientes y no excederse de 40, numerados de acuerdo al texto, según las indicaciones del Comité Internacional de Editores de Revistas Médicas. Ejemplos:
   a. Artículo ordinario de revistas: Melchor JL., Gracida C,I Cancino J, San Martín MA.: Primera experiencia a largo plazo en México con donadores asistólicos: Reporte de 12 casos, Nefrología Mexicana., 2001, 22 (7):21-24.
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   c. Comités o grupos de investigación como autores: The Mycophenolate mofetil, acute renal rejection study group: The years foloww-up, Trasplantation, 2001;71:1091-1097.

ARTÍCULO ORIGINAL

1. **Página inicial**
2. **Resumen** estructurado con subtítulos con 150 palabras máximo, resultados estadísticos, hallazgos y conclusiones. Al final del resumen se escriben palabras clave de 3 a 5 (que ayudan a los indicadores a clasificar el artículo) Objetivo: Material y métodos, estadística y conclusiones.
3. **Resúmenes en inglés:** una traducción del anterior clara y precisa. (3 a 5 Keywords)
4. **Texto del trabajo:**
   a. **Introducción:** en un máximo de dos páginas, explique el propósito, los antecedentes y la definición del tema principal. Con comentarios breves sobre lo que se quiera hacer énfasis en material y métodos. No incluya al autor ni señale conclusiones del trabajo realizado, no agregue tablas o figuras en esta sección.
   b. **Material y métodos:** Debe describirse claramente la forma como se seleccionaron los sujetos de observación o de experimentación.

Tratándose de experimentos en seres humanos, debe mencionarse la aceptación del enfermo y del comité de Ética Institucional que supervisa el seguimiento de las normas internacionales de la Declaración de Helsinki de 1983. No se usen nombres, iniciales o números de claves hospitalarias de los pacientes, ni fotografías completas de la cara (deben cubrirse los ojos) o de otras zonas que puedan identificarlos.

Identifique los métodos de estudio y los procedimientos utilizados para que se puedan reproducir por otros investigadores. Los métodos conocidos se pueden referir brevemente, con citas bibliográficas de los autores. Señale los medicamentos y productos químicos utilizando sus nombres genéricos, indicando dosis y modo de administración.

Indique el tipo de recolección de datos clínicos: prospectivo, retrospectivo, aleatorio, doble ciego, etc.

Señale el método estadístico utilizado.

   c. **Resultados.** Deben presentarse en forma ordenada, lo más precisa y completa posible.

Los datos obtenidos no deben interpretarse o valorarse en esta sección.

No deben emplearse expresiones verbales cuantitativas como: rara, frecuentemente, ocasionalmente, la mayoría, no utilizar pronombres personales como: "yo" o "nosotros" sino el termino neutro "se encontró", se obtuvo. Siempre sea posible utilizar resultados matemáticos, vgr. 5.7% y datos numéricos de significación estadística: $p < 0.01$.

Las tablas y gráficas anexas solo las más indispensables para enfatizar los resultados obtenidos. No repetir en el texto los datos de las tablas, ni hacer tablas con datos fácilmente deducibles de texto.

   d. **Discusión.** Inicie la redacción de esta sección señalando aspectos nuevos e importantes del estudio realizado, no repita datos o información ya presentados en introducción o resultados.

Compare sus resultados con los investigadores recientes

sobre el tema. Señale las limitaciones del problema y sus consecuencias para orientar investigaciones futuras. Si desea proponer hipótesis, exprese con claridad que no son conclusiones sino posibilidades.

e. **Conclusiones.** Las conclusiones señaladas deben ser breves y estar respaldadas por los datos recolectados. No enliste demasiadas conclusiones.
f. **Referencias** ya señalado.

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La revisión de un tema, y particularmente, la revisión "a fondo" es una parte importante de las publicaciones medicas porque sintetizan lo publicado previamente y actualizan los progresos observados en el estudio y tratamiento de los problemas renales. El tema debe ser de interés general, escrito preferentemente por un experto.

1. **Página inicial**
2. **Resumen en inglés y español** con "palabras clave".
3. **Texto.** Debe ser claro, preciso, siguiendo el orden estructurado. Algunos artículos con términos poco usados o de reciente adquisición pueden requerir un "glosario" en una hoja aparte, así titulada.
4. **Conclusiones:** sirven para enfatizar la tendencia actual en los resultados
5. **Referencias bibliográficas:** las mismas indicaciones. Se recomiendan de 50 a 80 referencias, la mitad cuando menos de los últimos 5 años.

CASO CLINICO

1. **Página inicial:** organizada en la misma forma
2. **Resumen** breve en español e inglés con palabras claves y una introducción limitada a los antecedentes en la literatura, frecuencia de presentación y terapéutica actual.
   Anotar 3 secciones principales: Introducción, Descripción del o de los casos y Discusión, referencias bibliográficas no más de 10.
3. **Resumen en inglés**
4. **Introducción** (opcional)
5. **Presentación del caso o casos:** descripción completa de los hallazgos clínicos y paraclínicos.
6. **Discusión:** debe de iniciarse con el desarrollo del caso desarrollando argumentos sustentables en la etiopatogenia del problema, demostraciones basadas en evidencias. Si se disponen de estudios histopatológicos, se harán las co-relaciones clínico patológicas. Si el paciente ha fallecido se investigará el protocolo de autopsia, si es posible.
7. **Referencias bibliográficas:** se escogerán 10 o menos referencias aplicables al caso siguiendo, los procedimientos especificados.

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Son aquellos que no entran en alguna de las clasificaciones previas, pero por su importancia son susceptibles de publicación.

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.Es un documento con comentarios críticos sobre algún material publicado en la propia revista, el cual tendrá por objetivo el aclarar hechos o circunstancias contenidas en dicho material, o bien para inquirir sobre conceptos confusos.
También es posible que trate acerca de temas de importancia para la institución de la revista. La extensión máxima será de tres páginas tamaño carta, incluida la bibliografía, e idealmente no deberá contener figuras ni tablas.

PREPARACIÓN DE MANUSCRITOS

Formato del manuscrito

Los manuscritos deben adherirse a disposición estándar y las directrices de la extensión del texto. Los manuscritos deberán presentarse en Microsoft Word, a doble espacio utilizando fuente en tamaño 12 (preferentemente Times New Roman) y los márgenes justificados. Las páginas deben estar numeradas empezando por la del título en español e inglés. Identificar el nombre del autor para correspondencia. Se pide NO ENVIAR MANUSCRITOS en formato PDF. Los manuscritos no deben presentarse en control de cambios. Todos los valores de parámetros bioquímicos deben expresarse en unidades convencionales (Sistema métrico decimal SMD). Si es necesario, el Sistema Internacional de Unidades (unidades SI) puede ser colocado dentro de un paréntesis, inmediatamente después de las unidades de convenciones del SMD. Tablas de conversión están disponibles en JAMA 1986; 255 (17): 2329-2339 o Ann Intern Med 1987; 106 (1): 114-129.

PÁGINA DEL TÍTULO

La página del título debe de incluir:
1. Título del manuscrito en español e inglés.
2. Los nombres y grados de cada autor.
3. El título actual y la afiliación de los autores.
4. Datos del autor para correspondencia: nombre, dirección, teléfono, números de fax y correo electrónico del autor.
5. Título corto: (45 caracteres o menos incluyendo espacios) para utilizarse en el encabezado de la página.

AUTORÍA

Todas las personas listadas como autores deben de seguir los criterios de paternidad literaria. Cada autor debe participar lo suficiente en el trabajo para tomar responsabilidad del contenido del documento y aprobar la versión final del manuscrito. La paternidad literaria se debe basar en las diferentes contribuciones que puede tener cada uno de los autores en:

- Diseño del estudio.
- Generación, recolección, análisis e interpretación de los datos.
- Redacción y/o revisión del manuscrito.
- Aprobación de la versión final de manuscrito.

El autor que sea nombrado para recibir la correspondencia del manuscrito, sus datos deben aparecer en la página del título y debe ser el mismo que envíe el manuscrito con nuestro Director-editor

RESUMEN

La página del resumen incluye el título del manuscrito pero no otra información de identificación. En el caso de investigación y artículo original se debe realizar un resumen estructurado utilizando los siguientes títulos.

Introducción: breve.

Objetivo: declarar de forma clara el propósito del estudio.

Material y métodos: diseño: diseño de la investigación. **Sujetos:** datos demográficos, criterios de selección y grupo control. **Intervención:** metodología y descripción del tratamiento utilizado. **Principal resultado medido:** variable utilizada para evaluar el efecto de la intervención.

Resultados: hallazgos principales del estudio.

Conclusiones: breve resumen de los resultados que son directamente apoyados por la evidencia.

El límite del resumen es de 300 palabras o menos. Al final del resumen, colocar una lista de cinco palabras claves.

ABREVIACIONES Y ACRÓNIMOS

Los términos complejos utilizados frecuentemente en el manuscrito deberán ser abreviados. Las abreviaciones deberán ser colocadas entre paréntesis la primera vez que se utilicen en el resumen y nuevamente la primera vez que se utilicen en el texto. No utilizar abreviaciones y acrónimos en el título.

FORMATO DE FUENTES DE FINANCIAMIENTO

Listar fuentes de financiamiento en una manera estándar para facilitar el cumplimiento de los requisitos de los financiadores. Ejemplos:

Financiamiento: Este trabajo es apoyado por el Instituto Nacional de Salud (números de convenio si aplica); Fundación América Latina para la paz, México, D.F., y los Institutos Nacionales de Salud (números de convenio). Cuando un financiamiento es una donación o recursos disponibles por parte de la universidad, colegio, instituto de investigación, agregar el nombre de la institución u organización que proporciono ese financiamiento.

No es necesario incluir descripciones a detalle del programa o el tipo de donaciones o premios.

Si no se dio ningún tipo de financiamiento a la investigación agregar el enunciado: “Esta investigación no recibió ninguna donación del sectores público o comercial”, o bien “sin fines de lucro”.

FIGURAS Y CUADROS (tablas)

Figuras

Llamaremos figura a: ilustraciones, dibujos, gráficas y fotografías. Las ilustraciones, dibujos y gráficas deben ser realizados en computadora. Las figuras deberán ser numeradas consecutivamente de acuerdo al orden en el que aparezcan en el texto (por ejemplo, Figura 1, Figura 2, Figura 3, etc.). Las figuras deberán adjuntarse en archivos separados y no estar integradas en el archivo del manuscrito.

Para las figuras solo aceptamos en formato JPG en resolución mínima de 150 pixeles, en otro formato o menor resolución, no son aceptables, porque son de mala calidad (baja resolución). Se pide envíen las figuras de preferencia en color junto a su archivo del texto.

Cudaros (tablas)

Incluir un título para cada cuadro. Numerar los cuadros consecutivamente en el manuscrito (Cuadro 1, Cuadro 2, Cuadro 3, etc.). Utilice símbolos estandarizados de formato superíndice (*, †, ‡, § ...) para citar o especificar algo del cuadro. Los autores deben colocar las notas abajo de la cuadro, en orden, leyendo de izquierda a derecha y de arriba hacia abajo. Deben comenzar una nueva serie de notas debajo de cada cuadro.

Si algún cuadro o figura ha sido publicada con

anterioridad, se debe enviar junto con el manuscrito la copia de la CARTA DE PERMISO del propietario de los derechos de autor. Se debe dar reconocimiento a la fuente original en el cuadro o figura, anotando la referencia completa en la sección de Referencia del manuscrito. La leyenda de la figura (o notas al pie del cuadro) deben concluir con: "Reproducido con permiso", seguido por el correspondiente número de referencia. Los autores son responsables de obtener la autorización y derechos para imprimir y publicar vía electrónica dichas figuras y cuadros. Los autores son responsables de realizar el pago correspondiente en su caso para la obtención de dichos permisos.

DECLARACIÓN DE CONFLICTO DE INTERÉS FINANCIERO Y APOYO

Reconocer el apoyo que tuvo la investigación por parte de fundaciones o industria y revelar cualquier potencial conflicto de interés financiero. Deberá ser declarada cualquier afiliación y/o relación significativa con cualquier organización o entidad que tenga un interés financiero directo o indirecto, ejemplo: empleo, consultas, subsidios, honorarios. Las especificaciones dentro de la declaración permanecerán de forma confidencial.

Los editores pueden solicitar a los autores del estudio que tengan un conflicto de interés, se agregue una declaración que diga: "Tengo acceso completo a los datos de este estudio y tomo la completa responsabilidad de la integridad de los datos así como de la exactitud en el análisis de los mismos". Si el Editor considera apropiado realizar una declaración general al respecto, ésta se agregará en el apartado de "Reconocimiento" del manuscrito. La sección de Reconocimiento debe revelar todas las fuentes de apoyo para el trabajo, tanto financieras como materiales. Si no hay conflicto de interés financiero identificado, se debe escribir seguido del nombre del autor.

REFERENCIAS

Las referencias deben ser numeradas según el orden de aparición en el texto, mediante números en formato superíndices. Las referencias deben ser compiladas al final del manuscrito de acuerdo con el orden de citación en el texto y deben seguir el estilo y el formato de la Asociación Médica Americana (AMA por sus siglas en inglés).

Los autores que utilizan software de referencia, tales como EndNote o Reference Manager deben seleccionar estilo NLM/PubMed.

Las referencias deben escribir a doble espacio en una página separada al final del manuscrito. Al momento de citar, se deben abreviar los nombres de las revistas como se marca en PubMed. Listar hasta seis autores y/o editores, si hay más de seis autores se listaran sólo los tres primeros seguido de la leyenda "*et al*." En el caso de citar a las revistas, se deberá incluir el volumen seguido del número de la revista entre paréntesis. La exactitud de la información de referencia es responsabilidad del autor. Indicar si la fuente es un artículo completo, resumen o libro; en caso de artículos, señalar todas las páginas que abarca el artículo. Toda la información de referencia debe ser completa cuando se envíe el manuscrito.

Ejemplos de referencias:

Artículo de revista: seis o menos autores:
Eyre S, Attman P, Haraldsson B. Positive effects of protein restriction in patients with chronic kidney disease. J Ren Nutr. 2008;18(3):269-280.

Artículo de revista: más de seis autores:
Fernández-Reyes MJ, Sanchez R, Garcýa L, *et al.* Acute responses of gastrointestinal hormones to both oral and parenteral intradialytic nutrition. Am J Nephrol. 2010; 32(3): 272-278.

Artículo de revista en procesos de impresión:
Steiber AL, Kopple JD. Vitamin status and needs for people with stage 3-5 chronic kidney disease. J Ren Nutr. (in press)

Libro Completo.
Byham-Gray LD, Burrowes JD, Chertow GM, eds. Nutrition in Kidney Disease. Totowa, NJ: Humana Press; 2008.

Capítulo del libro:
Wilkens KG, Juneja V. Medical nutrition therapy for renal disorders. In: Mahan LK, Escott-Stump S, eds. Krause's Food & Nutrition Therapy. 12th ed. St. Louis, MO: Saunders; 2008:921-958.

Suplemento de un artículo de revista:
Gullett NP, Hebbar G, Ziegler TR. Update on clinical trials of growth factors and anabolic steroids in cachexia and wasting. Am J Clin Nutr. 2010;91(4)(suppl):S1143-S1147.

Resumen de PubMed:
Szklarek-Kubicka M, Fijalkowska-Morawska J, Zaremba- Drobnik D, Ucinski A, Czekalski S, Nowicki M. Effect of intradialytic intravenous administration of omega-3 fatty acids on nutritional status and inflammatory response in hemodialysis patients: a pilot study [abstract]. J Ren Nutr. 2009; 19(6): 487-93. http://www.ncbi.nlm.nih.gov/pubmed/19616450. Consultado en Diciembre 24, 2010. PMID: 19616450.

Editorial:

McCarron DA, Drueke TB, Stricker EM. Science trumps politics: urinary sodium data challenge US dietary sodium guideline [editorial]. Am J Clin Nutr. 2010;92(5):1005-1006.

Epub ya disponible:

Kagoma YK, Weir A, Iansavichus AV, *et al*. Impact of estimated GFR reporting on patients, clinicians, and health-care systems: a systematic review [publicado en línea cerca de imprimirse Diciembre 9 2010]. Am J Kidney Dis. 2010. http://www.ncbi.nlm.nih.gov/pubmed/21146269. Consultado en Diciembre 24, 2010.

ENVÍO DE MANUSCRITOS

El proceso de envío de los manuscritos para revisión en Word al correo electrónico; revistasomtox@somtox.com.mx, donde le será enviado un número único de registro, para dar seguimiento. El Comité editorial le enviará por correo una carta al autor corresponsal acusando de recibido. Toda aceptación de manuscritos está sujeta a revisión editorial.

PROCESO EDITORIAL Y DE ARBITRAJE DE PARES

Todos los manuscritos se envían a revisión. Cada manuscrito es asignado a un editor y/o coeditor que tienen experiencia en la materia, toma de decisiones tempranas sobre el manuscrito. Después de la revisión por el editor, los manuscritos cuya escritura no sea clara, la información no sea importante o de interés para la audiencia de la revista serán rechazados en esta etapa, por el contrario si el manuscrito se juzga que es adecuado y competitivo para su publicación en la revista, se pasa al proceso de arbitraje.

En el proceso de arbitraje, los manuscritos serán enviados a expertos por pares. El proceso de revisión es de "doble ciego" para que las identidades de los autores y de los árbitros no sean reveladas entre ellos. Los revisores evalúan cada artículo sobre la base del contenido, la originalidad, el rigor científico, claridad y contribución al campo de urgencias. El proceso de revisión toma alrededor de 5 semanas. Los revisores proporcionan comentarios para el editor y para los autores. Cambios en el estilo y la claridad del documento se hacen a discreción de los colaboradores. Todos los cambios sustanciales requerirán de la aprobación del autor. Después de la revisión (por lo general de tres a cinco semanas después de la fecha del envío a los revisores), se le notificará al autor corresponsal si el manuscrito ha sido aceptado, requiere revisión o es rechazado. Los manuscritos aceptados serán editados de acuerdo al formato de estilo de la revista y regresados al autor para aprobación de la versión final.

Los autores son responsables de todas las afirmaciones realizadas en el trabajo.

EDICIÓN

Los manuscritos son editados acorde al estilo de la revista. Si alguna sección del manuscrito en particular no es clara o requiere información adicional, el editor de textos le hará preguntas al autor. Estas preguntas o consultas al autor, van a aparecer en los márgenes de las correcciones que se envían al autor. Los cambios realizados en el estilo, gramática y redacción/legibilidad no deben ser alteradas por el autor a menos que se trate de un error científico.

OBSERVACIONES

El manuscrito debe enviarse, a la "**Sociedad Mexicana de Toxicología**", junto con una carta del autor responsable del trabajo y colaboradores, cediendo sus derechos editoriales a la Revista (copyright) y asegurando que el artículo no ha sido enviado a publicación previamente. Con nombre y firma de todos los autores. Esta carta deberá enviarla junto con su artículo, al siguiente correo:

revistasomtox@somtox.com.mx