

# ETIOLOGÍA DEL TRASTORNO DE HIPERACTIVIDAD: UNA REVISIÓN BIBLIOGRÁFICA DEL PERÍODO 1999-2002

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## **INTRODUCCIÓN.**

Nos parece importante recoger el significado del término *etiología* asignado tanto en el *Diccionario de la Lengua Española* de la Real Academia Española como en el *Diccionario Terminológico de Ciencias Médicas*: “parte de la medicina que tiene por objeto el estudio de las causas de las enfermedades”. En esta definición se desliza un determinado posicionamiento que pudiera condicionar nuestro acercamiento al trastorno.

## **Propósito:**

Este artículo lo realizamos en el marco del I Encuentro Virtual sobre el Trastorno por Hiperactividad, a desarrollar en la lista de correo de RedIris "Hiperactividad, Atención, Impulsividad" HIP-AT-IM @LISTSERV.REDIRIS.ES.

Nuestro propósito es ofrecer al foro el resultado de una revisión bibliográfica de las publicaciones científicas de los últimos tres años (plazo comprendido entre 2002 y 1999) que traten el tema de la etiología del Trastorno. Este trabajo lo consideramos como un punto de partida para otros de mayor profundización, que siempre lo podrán perfeccionar.

Inicialmente nos propusimos revisar los últimos 10 años, pero razones de tiempo y de intención de centrar la revisión en una época de avance de la neurología y la genética, nos llevaron a reducir el plazo a los tres últimos años.

## **METODOLOGÍA:**

Realizamos una búsqueda de referencias bibliográficas en la base de datos PSYCINFO, así como en MEDLINE y PUBMED, analizando sus abstracts.

## **RESULTADOS Y DISCUSIÓN:**

Obtuvimos inicialmente 260 referencias en la base PSYCINFO correspondientes al periodo 1992-2002, quedando reducidas a 126 referencias al centrarnos en el periodo 1999-2002. La comparación entre el número de años transcurridos en cada periodo y el número de publicaciones respectivas nos confirma el hecho del aumento imparable de la cantidad de investigaciones que se viene produciendo en proporción geométrica en muchos campos del saber, incluyendo el que aquí nos interesa; esto es, en los últimos tres años se ha producido la mitad de los artículos de todos los últimos diez años.

Otros estudios anteriores al nuestro han hecho revisiones bibliográficas, aunque en periodos distintos

y sin especificar las fuentes de tales referencias (Sandoval et al, 1999).

Algunas publicaciones dejan constancia de la confusión y contradicción existente en el abordaje de la etiología del Trastorno (Reeve et al, 2001; Hinshaw et al, 2001; Bain, 2001; Burt et al, 2001), indicando que algunas de ellas incluso caen dentro de la categoría de conjeturas (Johnsen, 2001).

La existencia de diferentes aportaciones y la contraposición entre algunas de ellas hacen resaltar la conclusión de la importancia y de la urgencia de precisar la definición del Trastorno:

- es posible que estemos hablando de diferentes “Trastornos de Hiperactividad”, que se correspondan a diferentes etiologías, como hipótesis a contrastar.
- muchas de estas investigaciones hacen hincapié en la frecuente comorbilidad de este Trastorno con otros (obsesivo compulsivo, negativista desafiante, de conducta, manía, tendencia a fumar, síndrome de Tourette) (Tercyak et al, 2002; Budman et al, 2000; Hill et al, 2000; Zohar et al, 1999).
- el propio trastorno es definido como “psiquiátrico”, como “neurológico” o como “psiconeurológico” dependiendo de la especialidad que lo estudia.
- la ética y la deontología científicas en el trato con seres humanos dificulta seriamente la aplicación de una metodología estrictamente experimental que nos permita hablar con rigor de causalidades, y obliga a las investigaciones a usar métodos correlacionales, cuyas características y limitaciones debiéramos tener bien presentes a la hora de formular conclusiones.
- tamaño pequeño de las muestras, falta de grupos de control, (Muglia et al, 2002; Reeve et al, 2001; Weller et al, 1999; Weller et al, 1999)

Las publicaciones revisadas se posicionan respecto a la posible etiología en un planteamiento único y algunas veces excluyente, ya sea biologicista o psicológico / psicosocial, o en un planteamiento mixto, integrador de ambos modelos.

Esta “Torre de Babel” acerca del Trastorno es tan evidente, y quizás tan generadora de confusión más que de construcción, que algunas publicaciones se centran específicamente en el estudio del fenómeno de dicha “Torre de Babel” (Garrett, 2000).

Las etiologías abordadas en los estudios incluyen las relacionadas con los siguientes aspectos:

**\* genéticos:**

no relación entre determinados genes y el TDAH en una muestra (Barr et al, 2001)  
gen receptor de la dopamina D3 (Muglia et al, 2002; Denney et al, 2001)  
causas genéticas directas, o mediante interacción con el ambiente (Nadder et al, 2002)  
diferencias entre la severidad del TDAH (Thapar et al, 2001; Willcutt et al, 2000)  
gen de la dopamina D2 (Sullivan et al, 2001)  
y su interacción con el sexo (Rhee, 2000)  
gen receptor de la dopamina D4 (Sunohara et al, 2000; Biederman et al, 2000; Faraone et al, 1999; Thapar et al, 1999)  
en relación con el síndrome de Tourette (Sheppard et al, 1999; Pauls et al, 1999)  
Soo et al (1999), con diferente importancia del ambiente según sexo  
Waldman et al, 2001; Todd, 2001; Asherson et al, 2000; Faraone, 2000; Kuntsi et al, 2000; Bradshaw et al, 2000; Biederman et al, 1999; Spencer, 2002; Todd et al, 2001; Rapport et al, 2000

**\* Disfunciones del Sistema Nervioso Central:**

desequilibrio entre los sistemas motores cerebrales lateral y medial (Archibald et al, 2002)  
receptores adrenérgicos Alfa1C y Alfa2C (Barr et al, 2001)

hipoactivación cortical y falta de maduración del SNC (Clarke et al, 2002)  
disfunción del lóbulo frontal (Reeve et al, 2001)  
rutas frontales-subcorticales (Spencer, 2002; Biederman et al, 2000)

modelo de lateralización cerebral (Ponseti et al, 2001)  
funciones hipocatecolamina en la región subcortical y en la cortical prefrontal en cuanto síndromes secundarios de déficit energético (Todd et al, 2001)  
sistema serotoninérgico (Shalev et al, 2001)  
implicación de varias zonas cerebrales (Bradley et al, 2001)  
núcleo caudado y su relación con la actividad física (Wendt, 2000)  
hipoactividad de las zonas cerebrales prefrontal, estriada y límbica (Rapport et al, 2000)  
Kalbfleisch, 2001; Kolar et al, 2000

**\* ambientales:**

factores culturales, ambientales y parentales (Ravenel et al, 2002)  
Faraone et al, 2000

**\* familiares:**

Thomas, 2000  
exposición prenatal a cigarrillos y alcohol (Hill et al, 2000)  
factores perinatales (Zohar et al, 1999)

**\* infecciosos:**

de los ganglios basales (Yaryura et al, 2001)

**\* de corte psicoanalítico:**

problemática del funcionamiento del ego (Gilmore, 2000)  
disociación de partes del self (Low, 1999)

**\* Autores que tienen en cuenta tanto el modelo biológico como el psicológico:**

Gerjets et al, 2002; Amador et al, 2001; Hinshaw et al, 2001; Dinter, 2001; Bain, 2001; Solanto, 2001; Rapport, 2001; Faraone et al, 2001; Bradley et al, 2001; Gilmore, 2000; Martens, 2000; Kuntsi et al, 2000; Champbell, 2000; Zohar et al, 1999; Cook, 1999; Kotasova, 1999

Muy importante es el dato aportado por Baumeister et al (2001) sobre la inconsistencia entre los estudios que apoyan la etiología biológica del Trastorno y por Faraone et al (1999) en el mismo sentido relacionado con el papel del gen de la dopamina D-sub-4.

Finalmente, algunas obras que nos parecen interesantes son

- la tesis de Memenamy (2001) en la que generó un diseño para evaluar las atribuciones que los niños realizaban sobre la etiología del Trastorno, así como el estudio de Tingle (2000) en que sometía a grupos familiares a educación acerca del TDAH y observaba los cambios en los esquemas sobre etiología en los niños y las niñas, así como en los padres y las madres de forma diferenciada.
- el estudio longitudinal realizado por Loeber et al (2000) durante 13 años.
- los estudios de Jensen (2000) y de Samudra et al (1999) donde se analizan diferentes factores que pueden estar detrás del TDAH (familiares, genéticos, prenatales o perinatales, toxinas químicas, estresores psicosociales, estructuras cerebrales, disfunciones cerebrales).

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