LINEE GUIDA PER LA SCRITTURA DELLA TESI DI LAUREA BIOMEDICHE

Adriano Angelucci
2011

LE TESI

TESI COMPILATIVA

Riassunto critico delle informazioni presenti su un determinato argomento

TESI SPERIMENTALE

Esposizione dei risultati ottenuti tramite il proprio lavoro sperimentale

TESI SCIENTIFICA

1-L'oggetto della ricerca deve essere definito in modo che sia facilmente individuabile

di che si parla?

2-La ricerca deve essere originale

è la prima volta che se ne parla

3-La ricerca deve essere utile agli altri

parlarne può essere interessante

4-La ricerca deve fornire gli elementi per la verifica delle ipotesi che presenta

i risultati sono condivisibili

TESI SCIENTIFICA

4- i risultati sono condivisibili?

Tramite l'inserimento delle note bibliografiche:

Si compone delle citazioni nel testo e dall'elenco delle pubblicazioni citate

- le citazioni devono essere facilmente associabili all'informazione che è stata riportata

- l'elenco bibliografico deve permettere di rintracciare in maniera univoca la fonte

BIBLIOGRAFIA

CITAZIONI

Esistono due principali metodi per inserire le citazioni:

1- con numerazione progressiva nel testo tra parentesi tonde, quadre (1)...(2, 3)...(4-7)..., apice

2-con il nome del primo autore e l'anno di pubblicazione (Angelucci et al. 1999). In questo caso l'elenco bibliografico sarà in ordine alfabetico. (+ semplice)

ESEMPI DI BIBLIOGRAFIA

normal cells in their biochemical metabolism. One of the most prominent metabolic alterations in cancer cells is the increase in aerobic glycolysis and the dependency on glycolytic pathway for ATP generation, known as the Warburg effect (Warburg *et al.*, 1924; Warburg, 1930, 1956). As this metabolic alteration is frequently seen in

Warburg O. (1930). *The Metabolism of Tumors*. Costable: London.

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Warburg O, Posener K, Negelein E. (1924). *Biochem Z* **152**: 309–344.

ment: pathogen-associated molecular patterns (PAMPs) and ‘danger signals’ derived from host cell stress or death¹.

A growing body of research shows that a third class of stimulus, in the form of neural and endocrine signals that result from macroenvironmental sensing, also plays a significant role in modulating immune responses². In addition, immune mediators

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BIBLIOGRAFIA

ELENCO BIBLIOGRAFICO

Non esiste una formattazione standard ma diversi stili

Stile Vancouver:

articolo

libro

Citare risorse elettroniche

>rivista elettronica

Chappell, C. (2003). Researching vocational education and training: where to from here?. *Journal of Vocational Education and Training*, 55 (1), pp. 21-32, retrieved November 25, 2003, from <http://www.triangle.co.uk/vae/>.

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Kenneth, I. A. (2000). A Buddhist response to the nature of human rights. *Journal of Buddhist Ethics*, 8(4). Retrieved February 20, 2001, from <http://www.cac.psu.edu/jbe/twocont.html>

>Newsgroup, forum o discussion group

Frook, B. D. (1999, July 23). New inventions in the cyberworld of toylandia [Msg 25]. Message posted to <http://groups.earthlink.com/forum/messages/00025.html>

TESI COMPILATIVA: Struttura

Indice

Lista delle abbreviazioni

Introduzione

Corpo della tesi (paragrafi)

Conclusioni

Bibliografia

Appendici (eventuali)

TESI COMPILATIVA: Percorso

TESI COMPILATIVA: Consigli

Non è necessario partire dal primo capitolo

Farsi un'idea generale prima di iniziare a scrivere

Cercare di leggere direttamente le fonti bibliografiche

Usare fonti il più possibile aggiornate

Non spiegate dov'è Roma senza poi spiegare dov'è Timbuctu.

TESI COMPILATIVA: L'inizio

Oncogene (2006) 25, 4633–4646

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www.nature.com/onc

REVIEW

Glycolysis inhibition for anticancer treatment

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Weissman, D. **Tk1xA is an endogenous ligand for Toll-like receptor 3.** *J. Biol. Chem.* **279**, 12542–12550 (2004).

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Articolo sperimentale

0021-972X/01/\$03.00/0

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Leptin Is Associated with Increased Prostate Cancer Risk: A Nested Case-Referent Study*

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ABSTRACT

A Western lifestyle has been implicated in the pathogenesis of prostate cancer. However, no clear association between obesity and prostate cancer has been shown. Leptin may stimulate prostate growth and angiogenesis, and receptors for leptin are present in the prostate. Leptin may, thus, be associated with increased risk of prostate cancer.

One hundred forty-nine men with prostate cancer were identified (together with 298 matched referents) who, before diagnosis, had participated in population-based health surveys in Northern Sweden. Blood pressure, body mass index, and use of tobacco were recorded. Leptin, insulin, insulin-like growth factor I (IGF-I), IGF-I-binding proteins 1–3, testosterone, and sex hormone-binding globulin were analyzed in stored samples. Their influences on prostate cancer were estimated by conditional logistic regression analysis. Prostate cancer specimens were investigated for immunoreactivity for the leptin receptor.

Relative risk (95% confidence intervals) estimates of prostate cancer over the quintiles of leptin were 1.0, 2.1 (1.1–4.1), 2.6 (1.4–4.8), 1.4 (0.7–2.7), and 1.6 (0.8–3.2). Adjustments for metabolic variables, testosterone, and IGF-I and its binding proteins did not attenuate this increased risk. Immunoreactivity for the leptin receptor was detected in normal, high-grade prostatic intraepithelial neoplasia lesions and malignant prostatic epithelium.

Moderately elevated plasma leptin concentrations are associated with later development of prostate cancer. This may be due to direct effects of leptin on prostatic intraepithelial neoplasia lesions, or to indirect actions through other mechanisms. A critical fat mass related to an interior milieu favorable for prostate cancer development seems to exist, because intermediate but not high leptin levels are related to prostate cancer risk. (*J Clin Endocrinol Metab* 86: 1341–1345, 2001)

MEN LIVING IN Western countries have a much higher incidence of prostate cancer than men in the Far East, and immigrating Asian men gradually adapt to the incidence in the West (1), strongly suggesting that lifestyle factors are important in the pathogenesis of prostate cancer. A Western lifestyle, encompassing a high caloric intake from an energy-dense diet and a sedentary lifestyle, resulting in a positive energy balance and often obesity, has consistently been implicated as a risk factor for prostate cancer in international comparisons and experimental studies (2–4). However, individually based epidemiological studies on prostate cancer and diet have overall shown weaker associations (2), and epidemiological studies on prostate cancer in relation to obesity and physical activity have shown little or no association (2, 5). Besides insulin-like growth factor I (IGF-I), which has been shown to be a risk marker for prostate cancer in several epidemiological studies (6–8), no biomarker associated with a Western lifestyle has been linked to prostate cancer risk.

Leptin, the adipocyte-derived hormone, regulates satiety and energy expenditure by conveying information to the brain about the size of energy stores (9), and leptin deficiency

is associated with gross overweight in humans (10). However, apart from rare cases of congenital leptin deficiency (10), circulating levels of leptin are elevated in obesity (11) and increases with high-energy food intake (12). Leptin has also been associated with features of the insulin resistance syndrome (13). High expression of the leptin receptor has been observed in the prostate (14), and circulating leptin levels increase in parallel with prostate growth at puberty in the rat (15). Interestingly, leptin stimulates angiogenesis (16). Our hypothesis was that increased plasma leptin levels are associated with the development of prostate cancer.

Subjects and Methods

Study populations

The two northernmost counties in Sweden (Västerbotten and Norrbotten) with a total population of 510,000 constitute 1 of 39 collaborating centers in the WHO MONICA (Monitoring of Trends and Determinants in Cardiovascular Disease) study (17). Population-based surveys were performed in 1986, 1990, and 1994, and men and women aged 25–74, selected by stratified randomization for age and sex, were recruited. A community intervention program concerning cardiovascular disease and diabetes prevention—The Västerbotten Intervention Program (VIP)—was launched in 1985 in one of the counties (*i.e.* Västerbotten with a total population of 260,000; Ref. 18). Men and women were invited to a health survey (the same design as the MONICA population surveys) at their primary health care center the year they became 30, 40, 50, and 60 yr of age.

Participants in both the MONICA and the VIP surveys were requested to donate a fasting blood sample to be stored at the Medical Biobank at Umeå University Hospital for future research. Between January 1, 1985, and March 31, 1999, 26,856 men participated in the VIP

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Results

Baseline characteristics and plasma leptin levels are shown in Table 1. There were no significant differences in means between cases and referents for leptin or other variables. Because blood samples had been collected well before time of diagnosis (median, 3.9 yr), we combined data of cases and referents for analysis of interrelationships between leptin and other variables (Table 2). Plasma leptin was strongly correlated to aberrations associated with the insulin resistance syndrome, notably high BMI and hyperinsulinemia. After adjustments for age, BMI, and insulin, a significant correlation with glucose remained. Furthermore, a strong inverse correlation between leptin on one side and testosterone and SHBG on the other remained. The

TABLE 2. Bivariate and partial correlation coefficients between leptin and selected variables

Adjusted for	–	Age/BMI/insulin
Age	0.04	
BMI	0.62 ^a	
Diastolic BP	0.23 ^a	0.09
Systolic BP	0.16 ^b	0.00
Cholesterol	–0.05	0.00
Insulin	0.53 ^a	
Fasting glucose	0.19 ^a	0.11 ^c
Postload glucose	0.30 ^a	0.18 ^b
Testosterone	–0.29 ^a	–0.20 ^a
SHBG	–0.29 ^a	–0.13 ^b
Total PSA	0.04	0.05
IGF-I	–0.08	–0.04
IGFBP-1	–0.38 ^a	–0.05
IGFBP-2	–0.47 ^a	0.18 ^c
IGFBP-3	0.04	0.05

^a $P < 0.001$.

^b $P < 0.01$.

^c $P < 0.05$

Discussion

This study, the first nested case referent study on leptin and cancer risk to the best of our knowledge, showed a significantly increased risk for prostate cancer in the intermediate range of circulating leptin. We also found immunoreactivity for leptin receptors in prostatic cancer specimens, with a strong expression in high-grade PIN lesions, a precursor lesion for manifest cancer (21). To date, only two small case referent studies have been published on a possible association between leptin and cancer [*i.e.* prostate cancer (22) and breast cancer (23), respectively]. None of these studies detected a significant association between leptin and cancer risk, consistent with our findings in the subgroup of men with a short follow-up time.

The prospective design of our study ensures that the samples from cases and referents were handled in an identical fashion and also minimizes the risk that alterations in leptin are an effect and not a cause of disease, which may have confounded earlier studies. Acute and chronic disease may, thus, influence leptin levels (24). In addition, there seems to be a specific interval of leptin levels associated with development of prostate cancer.

Leptin may promote cancer development through direct effects in the prostate. High leptin receptor messenger RNA levels have been demonstrated in the prostate (14). We now demonstrate the presence of immunoreactive leptin receptors in normal prostatic epithelial cells, in high-grade PIN, and in prostatic cancer cells. Leptin may be involved in the secondary sexual organ growth spurt during puberty (15), and it is, therefore, possible that leptin by direct cellular effects could influence the development and progression of prostate cancer.

Articolo 1

References

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→ Articolo 2

REF → Articolo 3

REF

Considerando una media di 30 citazioni per articolo
Seguendo le referenze di 3 livelli di ricerca

Si hanno 27000 articoli da selezionare

Criteri di scelta della bibliografia

Attendibilità:

parametro non trasparente

Impact Factor (IF) = numero di citazioni

Pertinenza:

Solo gli argomenti della scaletta

Completezza:

Evidenze coerenti e convincenti

Data:

Articoli più recenti o la prima segnalazione

TESI SPERIMENTALE: Struttura

CAMMINO CONCETTUALE PER LA PREPARAZIONE DELLA TESI

ORGANIZZAZIONE PRATICA

LISTA DELLE ABBREVIAZIONI

L'uso delle abbreviazioni rende più fluida la scrittura e la lettura

Il mondo scientifico è pieno di sigle

Tutte le abbreviazioni devono essere spiegate

Le abbreviazioni sono acronimi di termini inglesi

TGF- β = Fattore di crescita trasformante di tipo beta (Transforming Growth Factor-beta)

RISULTATI

- Le figure e/o le tabelle
- Leggende

La sequenza delle figure deve seguire per quanto possibile un filo logico

LEGGENDE E RISULTATI

La leggenda è una descrizione concisa ma esaustiva che permette di interpretare la figura senza discutere i dati

Il testo dei risultati contiene la descrizione di quali sono i dati riportati nelle figure e delle motivazioni che hanno portato alla realizzazione degli esperimenti compiuti.

INTRODUZIONE

Basi scientifiche che permettono la corretta interpretazione dei risultati

Da evitare l'inserimento di elementi non pertinenti

Selezionare gli argomenti da trattare in modo che costituiscano capitoli

Le fonti da cui si attinge devono essere sempre riportate in bibliografia a meno di citazioni di elementi ormai assodati

Partire dall'argomento più generale a quello più specifico

TESI SPERIMENTALE

Proprietà antitumorali del Tè verde

INTRODUZIONE:

CAP 1= Il Tè nella storia dell'umanità

CAP 2= Le sostanze presenti nel Tè verde

CAP 3=Il Tè come farmaco

CAP 4= Fitoterapia antitumorale

SCOPO DEL LAVORO

Ogni lavoro sperimentale mira alla dimostrazione di ipotesi

Nel descrivere lo scopo del lavoro dobbiamo individuare **l'ipotesi di partenza**

Bisogna tenere in considerazione il substrato scientifico di partenza (introduzione)

Privilegiare l'originalità

SCOPO DEL LAVORO

Approfondire le basi molecolari
delle evidenze epidemiologiche e sperimentali
che suggeriscono nell'uso del Tè verde
un'importante strategia antitumorale

MATERIALI E METODI

Descrizione tecnica dei protocolli usati per l'esecuzione degli esperimenti

Deve contenere tutte le informazioni utili al lettore per ripetere gli esperimenti mostrati

Western blotting

Le cellule sono state lisate con RIPA buffer (Tris HCl 50 mM pH 7.5, NaCl 150 mM, Nonidet P-40 1%, Sodio Deoxicolato 0.5%, SDS 0.1 %) con l'aggiunta degli inibitori delle proteasi. Le proteine sono state separate con elettroforesi su gel di poliacrilammide al 15% in presenza di Sodio-Dodecil-Solfato (SDS-PAGE 15%) e trasferite su membrana di nitrocellulosa. Successivamente la membrana è stata messa ad incubare con anticorpo primario per 1h a temperatura ambiente. Sono stati effettuati dei lavaggi e poi la membrana è stata messa ad incubare con anticorpo secondario coniugato con la perossidasi di rafano (Horseradish Peroxidase -HRP) per 1h a temperatura ambiente. Il complesso enzimatico della perossidasi catalizza la conversione del substrato ECL (Enhanced Chemiluminescence) in un composto in grado di emettere chemiluminescenza.

CONCLUSIONI

Le conclusioni devono contenere:

1-Breve riassunto delle basi scientifiche di partenza

2-Esposizione dei risultati più importanti ottenuti

3-Interpretazione dei risultati nel contesto scientifico di partenza

4-Sviluppi futuri derivanti dalla continuazione del lavoro sperimentale

CONCLUSIONI

Le sostanze presenti nel Tè verde sono state per molto tempo ritenute...

Noi abbiamo dimostrato che il flavonoide...

Tale risultato conferma che l'assunzione di...

Studi futuri permetteranno di dimostrare che anche in vivo...

A CHE E A CHI SERVE LA TESI DI LAUREA

A CHE

OBBLIGO ACCADEMICO
A CONOSCERE IL METODO SCIENTIFICO
AD APPRENDERE IL LAVORO DI LABORATORIO
A DIVULGARE IL LAVORO DEL PROPRIO LABORATORIO

A CHI

A SE STESSI
AL TUTORE E A TUTTO IL LABORATORIO
ALLA COMMISSIONE ESAMINATRICE
ALLA PROPRIA FAMIGLIA
AI PROPRI AMICI