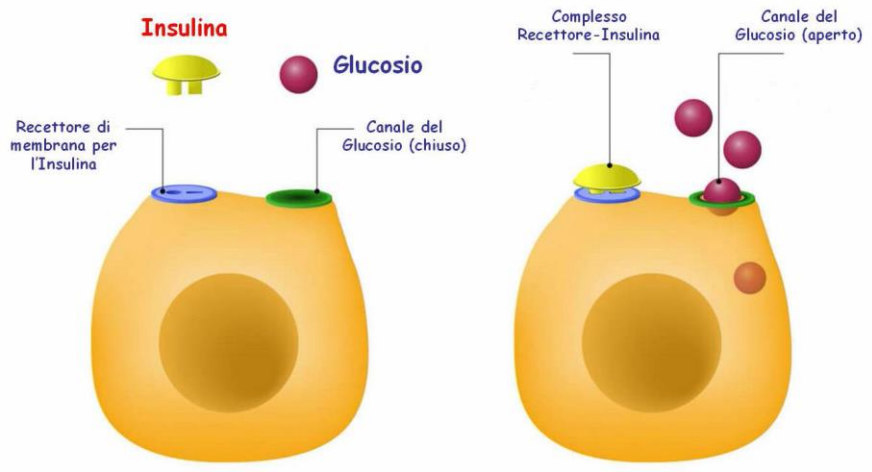
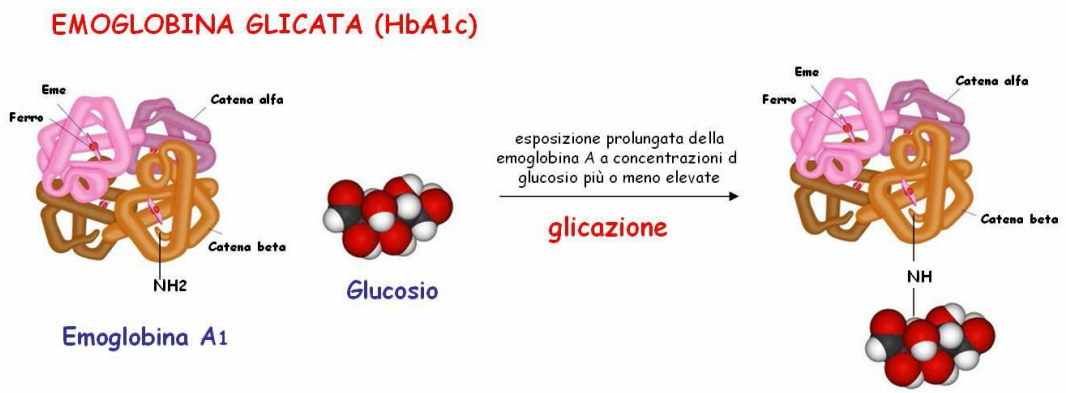
BIOLOGIA DI LABORATORIO PER LA NUTRIZIONE
Insulino-resistenza, Diabete e Marcatori di Metabolismo Glucidico

Enpab webinar



Mauro Mario Amato



Giovedì 9 Luglio 2020
ore 16.00 – 18.00

Ordine Nazionale dei Biologi

TEL. (06) 57.090.1 r.a. – Telefax: 57.090.235

00153 ROMA - Via Icilio, 7

www.onb.it segreteria@onb.it

Delibera del Consiglio
dell'Ordine Nazionale dei Biologi
n. 433 del 26 settembre 2019

Linee guida per la professione di Biologo Nutrizionista

6. DIVIETI SPECIFICI E LIMITI DELL'ESERCIZIO

Al professionista, nell'ambito dell'esercizio professionale, è fatto divieto di:

- ✓ effettuare diagnosi;
- ✓ prescrivere o consigliare terapie;
- ✓ prescrivere o consigliare farmaci;
- ✓ prescrivere o consigliare analisi cliniche;
- ✓ elaborare diete on-line;
- ✓ eseguire esami diagnostici nello studio professionale;
- ✓ vendere prodotti;
- ✓ utilizzare apparecchiature invasive e/o estranee all'attività professionale;
- ✓ utilizzare denominazioni professionali improprie.

In particolare, nell'ambito delle proprie competenze, il Biologo, per poter rilasciare un programma alimentare personalizzato, esegue una preliminare attività di anamnesi con la quale acquisisce puntuali indicazioni relative alle condizioni di salute, alla eventuale presenza di patologie certificate dal medico ed all'assunzione di eventuali farmaci, alle preferenze espresse dal cliente stesso, allo stile di vita ed alle abitudini alimentari.

Le analisi cliniche possono essere lette ed utilizzate per pianificare un programma alimentare personalizzato, senza in alcun modo configurare diagnosi di patologia.

In presenza di patologie riferite dal paziente e quindi in assenza di referti o anamnesi fisio-patologica del medico che ne comprovino la diagnosi, costituisce prassi corretta che il nutrizionista, richieda la collaborazione del medico di medicina generale, direttamente ovvero per il tramite dell'utente, al fine di ottenere una valutazione appropriata sullo stato di salute del paziente stesso. In attesa può comunque svolgere una attività di educazione alimentare.

Indagini di laboratorio — interpretazione → (no diagnosi) → **Strategia Alimentare opportuna**

Il Laboratorio nella gestione del METABOLISMO GLUCIDICO

Assetto Glicemico

dosaggio e valutazione di

Glicemia
Emoglobina HbA1c
Insulina
Microalbuminuria

si possono associare

PCR (Proteina C Reattiva)
OGTT (Curva da carico orale di glucosio)
Curva Insulinemica da carico orale di glucosio
Fruttosamine/Albumina glicata
Esame urine standard

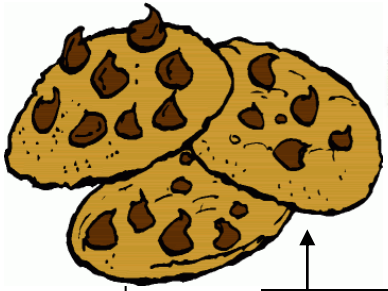
DIABETE

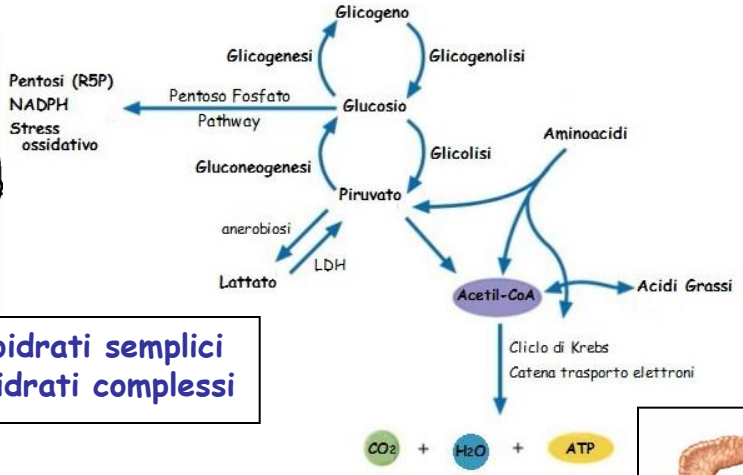
Mauro Mario Amato

METABOLISMO CARBOIDRATI

Mauro Mario Amato

Enpab webinar

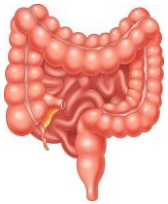




Carboidrati semplici
Carboidrati complessi

Amilasi salivare

amidi cotti → **Maltosio Maltotrioso Destrine**



Succo pancreatico → **Amilasi pancreatica**

Succo enterico → **Disaccaridasi**

Glucosio Fruttosio Galattosio

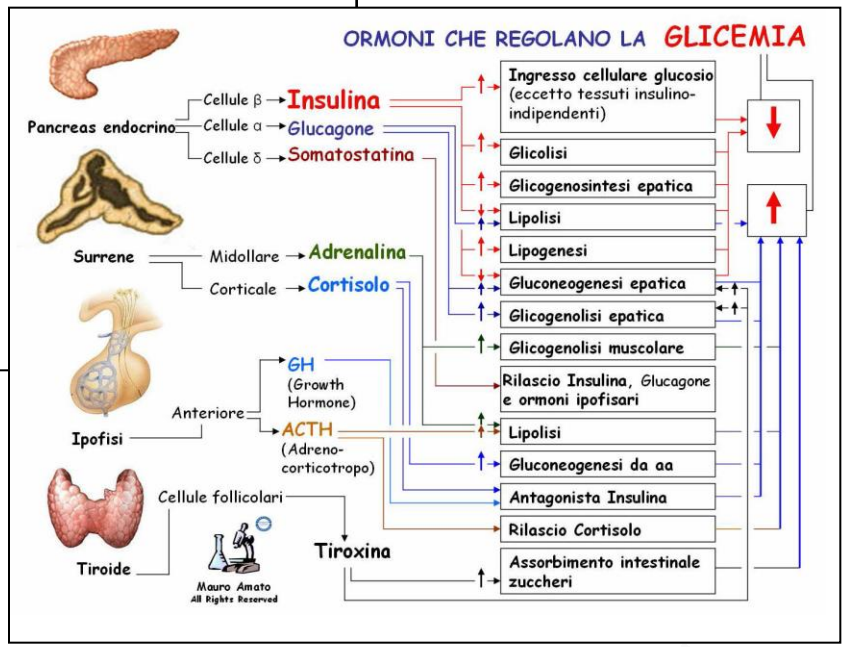
Glucosio

Glicogenosintesi

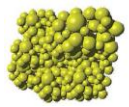
Glicogeno epatico

Glicogenolisi → **GLUCOSIO Ematico**

GLICEMIA (70 – 100 mg/dl)



Tessuto Adiposo

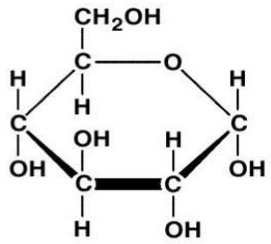


Tessuto Muscolare

Glicogeno muscolare

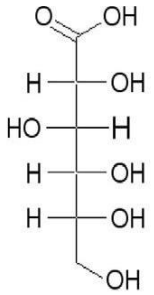
Dosaggio della GLICEMIA

Enzimatico-colorimetrico Trinder (GOD/POD)

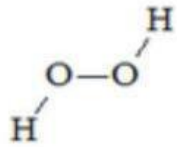


GOD

Glucosio ossidasi



\+



H_2O_2

POD

Substrato cromogeno

Composto colorato

Glucosio

Ac. Gluconico

Dosabile fotometricamente a determinate lungh.d'onda. L'intensità di colore è proporzionale alla quantità di **Glucosio** presente nel campione.

Metabolismo strettamente legato all'**Insulina**

Diminuzione concentrazione/attività insulina

Iperglicemia ⟶ **Diabete Mellito**.

- **Digiuno prolungato/Sforzo fisico/Errori congeniti metabolismo glucidico** ⟶ **Ipoglicemia**
- E' bene effettuare il **prelievo dopo 8-12 ore di digiuno** con il soggetto in esame che, durante i 3-4 gg precedenti la determinazione, abbia tenuto dieta e attività consueta.
- La glicemia può essere espressa in **mmoli/l** (**indicazioni IFCC**)
  Fattore di conversione 0.0555
  mg/dl x 0.0555 = mmoli/l

International Federation of Clinical Chemistry and Laboratory Medicine

ADA (American Diabetes Association) 2019

pre-diabete
100-125 mg/dl

cut-point per diabete
>125 mg/dl

ESC (European Society of Cardiology) 2019

cut-point per diabete
>109 mg/dl

IPERGLICEMIA

Valore di soglia oltre il quale si può parlare di iperglicemia:

> **126** mg/dl a digiuno.

sete

necessità di urinare più spesso

pelle secca

fame

assonnato

visione sfuocata

guarigione più lenta

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IPOGLICEMIA

Valore di soglia sotto il quale si può parlare di ipoglicemia:

70 - **60** mg/dl a digiuno

tremori

tachicardia

sudorazione

vertigini

ansietà

fame

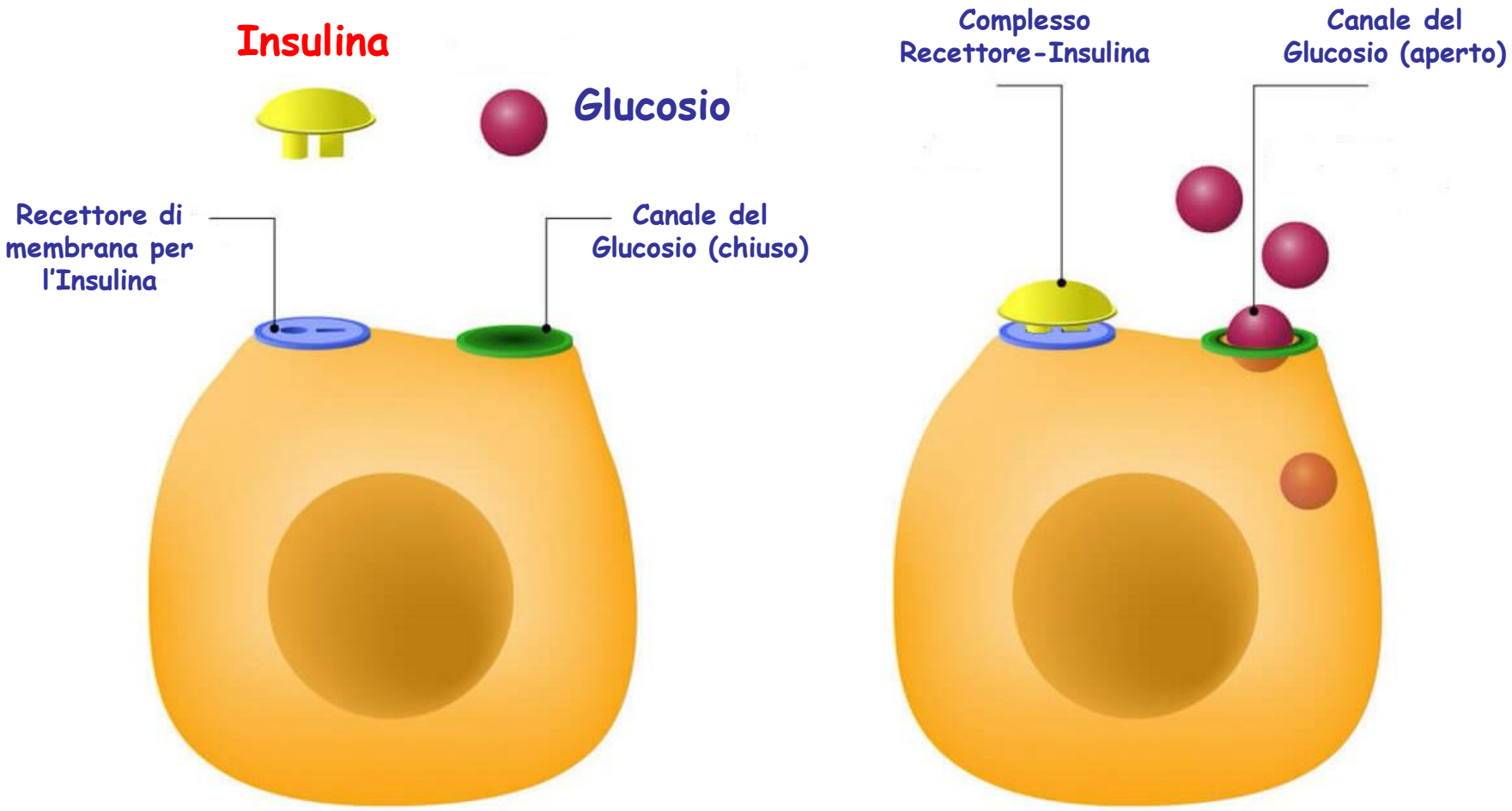
visione sfuocata

stanchezza

Mal di testa

arrabbiato

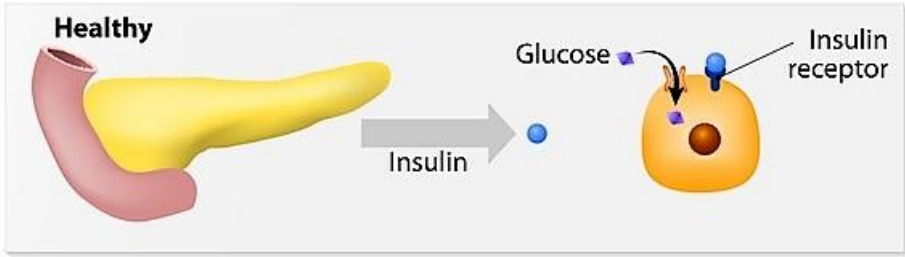
COME LAVORA L'INSULINA



Mauro Mario Amato

IL DIABETE MELLITO

Disordine metabolico con **iperglicemia cronica** e disturbi del metabolismo dei carboidrati, lipidi e proteine dovuta alla alterazione della secrezione/azione dell'**insulina**



DMT1 (Diabete Mellito Tipo 1)

- Distruzione cellule beta pancreas endocrino **su base autoimmune o idiopatica**. Di solito determina assoluta carenza di insulina.
- In genere **infanzia o adolescenza**.
- Variante **LADA** (Latent Autoimmune Diabetes in Adult).
- 5 – 10% diabetici

DMT2 (Diabete Mellito Tipo 2)

- **Deficit** parziale **di secrezione di insulina** progressiva nel tempo.
- **Insulino-resistenza** su base multifattoriale.
- Forma più **frequente nell'adulto**.
- Associazione con **obesità e altre malattie metaboliche**.

Diabete Gestazionale

- Diagnosticato per la **prima volta in gravidanza (2°/3° trimestre)** per difetti funzionali analoghi a DMT2.
- **Possono sviluppare DMT2** nel corso della vita (**30%**).

DMT3 Termine proposto per l'Alzheimer in seguito all'insulino-resistenza che si instaura a livello cerebrale
Non previsto nelle Linee Guida Diabete

Diabete Secondario

- Conseguente a patologie che interessano la secrezione o l'azione insulinica (pancretite cronica, pancreasectomia, ipercortisolismo) o all'uso cronico di farmaci (steroidi, antiretrovirali).

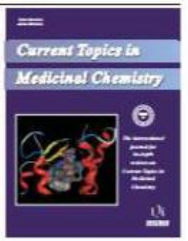
Diabete Monogenico (Diabete MODY)

- Modificazioni genetiche alteranti secrezione e/o azione insulinica.
- Maturity Onset Diabetes of the Young
  Forma rara di diabete (1-2%) non autoimmune caratterizzata da una iperglicemia familiare.

REVIEW ARTICLE

BENTHAM SCIENCE

Type 3 Diabetes Mellitus: A Novel Implication of Alzheimers Disease



Jerzy Leszek[1], Elżbieta Trypka[1], Vadim V. Tarasov[2], Ghulam Md Ashraf[3], and Gjumrakch Aliev[4,5,6,*]

DMT3 quale implicazione dell'Alzheimer

[1]*Department of Psychiatry, Wroclaw Medical University, Wroclaw, Poland;* [2]*Department of Pharmacology of Pharmaceutical Faculty, I.M. Sechenov First Moscow State Medical University, Moscow, Russia;* [3]*King Fahd Medical Research Center, King Abdulaziz University, Jeddah, Saudi Arabia;* [4]*GALLY International Biomedical Research Consulting LLC., 7733 Louis Pasteur Drive, #330, San Antonio, TX, 78229, USA;* [5]*School of Health Science and Healthcare Administration, University of Atlanta, E. Johns Crossing, #175, Johns Creek, GA, 30097, USA;* [6]*Institute of Physiologically Active Compounds Russian Academy of Sciences, Chernogolovka, 142432, Russia*

ARTICLE HISTORY

Received: April 29, 2016
Revised: November 01, 2016
Accepted: November 02, 2016

DOI: 110.2174/1568026617666170103163403

Abstract: The brain of patients with Alzheimer disease (AD) showed the evidence of reduced expression of insulin and neuronal insulin receptors, as compared with those of age-matched controls. This event gradually and certainly leads to a breakdown of the entire insulin-signaling pathway, which manifests insulin resistance. This in turn affects brain metabolism and cognitive functions, which are the best-documented abnormalities in AD. These observations led Dr. de la Monte and her colleagues to suggest that AD is actually a neuroendocrine disorder that resembles type 2 diabetes mellitus. The truth would be more complex with understanding the role of low-density lipoprotein receptor-related protein 1, Aβ derived diffusible ligands, and advanced glycation end products. However, now it known as "brain diabetes" and is called type 3 diabetes mellitus (T3DM). This review provides an overview of ***"brain diabetes"*** focusing on the reason why the phenomenon is called T3DM.

Keywords: Alzheimer disease, Diabetes mellitus, Insulin, Type 2 diabetes mellitus, Type 3 diabetes mellitus.

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https://www.ncbi.nlm.nih.gov/pubmed/28049395

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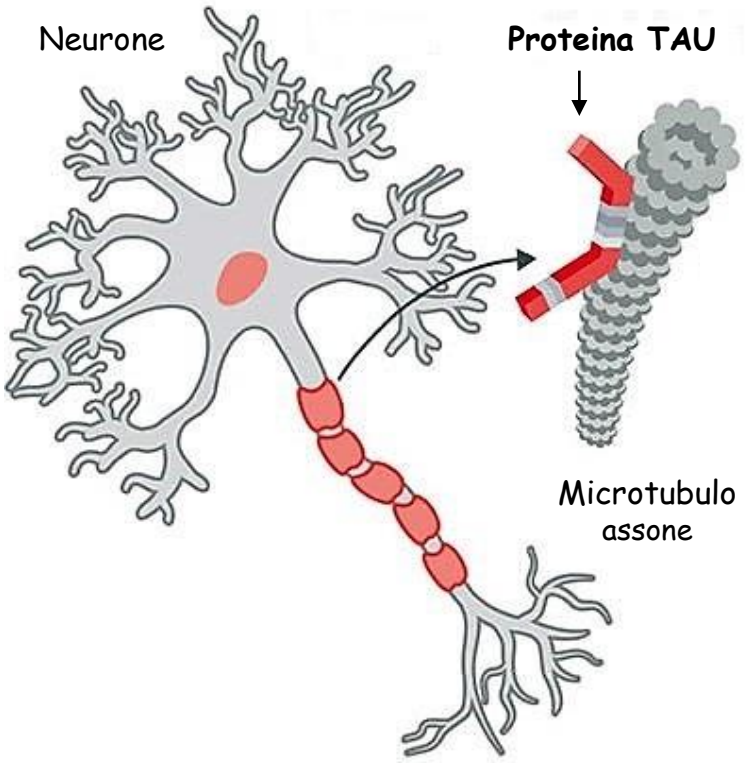
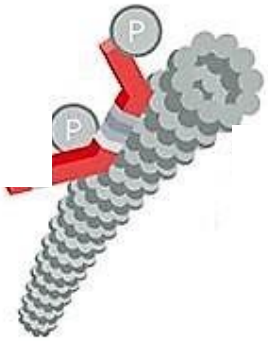
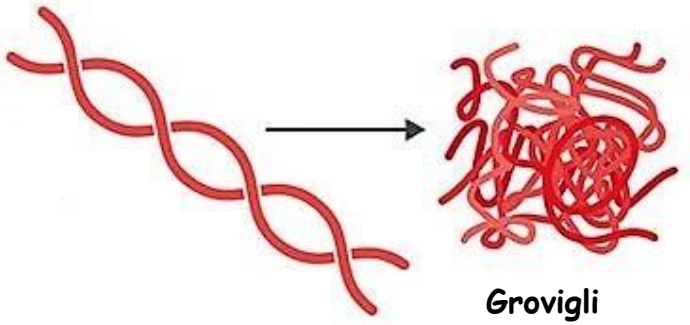
IPOTESI IPERFOSFORILAZIONE PROTEINA TAU





Iperfosforilazione
proteina TAU
(tau mutata)

Destabilizzazione microtubuli
Distacco componenti
(doppi filamenti ad elica)



Grovigli neurofibrillari TAU (NFT)

Alzheimer

DIABETE/RESISTENZA INSULINICA → ALZHEIMER → DIABETE MELLITO TIPO 3

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NFT
(grovigli neurofibrillari)

Fosforilazione proteina TAU

Resistenza insulinica/Resistenza IGF1
cerebrale

Utilizzo GLUT4 ↓

neurodegenerazione

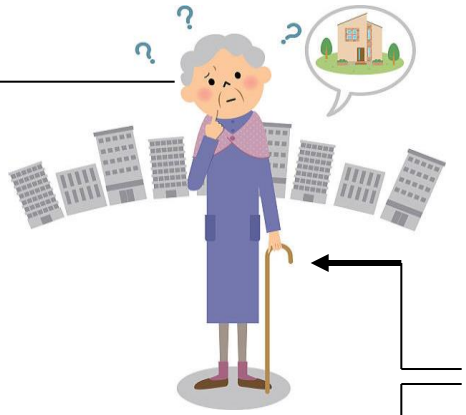
Apoptosi
Infiammazione

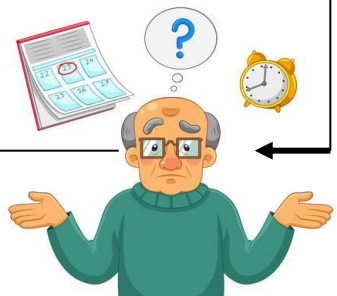
Accumulo ß-amiloide
Stress ossidativo
Fosforilazione proteina Tau
Apoptosi neuronale

Alzheimer

Stress Ossidativo

ROS
Danni DNA
Disfunzione mitocondri





Resistenza insulinica
cerebrale

DIABETE
MELLITO TIPO 3
(DMT3)

rischio ↑

Resistenza insulinica
periferica

Published in final edited form as:
Biochim Biophys Acta. 2017 May ; 1863(5): 1078–1089. doi:10.1016/j.bbadis.2016.08.018.

Is Alzheimer's disease a Type 3 Diabetes? A Critical Appraisal

Ramesk Kandimalla[1], **Vani Thirumala**[1,2], and **P. Hemachandra Reddy**[1,3]

[1]Garrison Institute on Aging, Texas Tech University Health Sciences Center, 3601 4th Street, MS 9424, Lubbock, TX 79430, United States

[2]BSA Neuroscience, University of Texas at Austin, Austin, Texas, USA 78712

[3]Department of Cell Biology & Biochemistry, Neuroscience & Pharmacology and Neurology Texas Tech University Health Sciences Center, 3601 4th Street, MS 9424, Lubbock, TX 79430, United States

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5344773/

Abstract

Recently researchers proposed the term 'Type-3-Diabetes' for Alzheimer's disease (AD) because of the shared molecular and cellular features among Type-1-Diabetes, Type-2-Diabetes and insulin resistance associated with memory deficits and cognitive decline in elderly individuals. Recent clinical and basic studies on patients with diabetes and AD revealed previously unreported cellular and pathological among diabetes, insulin resistance and AD. These studies are also strengthened by various basic biological studies that decipher the effects of insulin in the pathology of AD through cellular and molecular mechanisms. For instance, insulin is involved in the activation of glycogen synthase kinase 3β, which in turn causes phosphorylation of tau, which involved in the formation of neurofibrillary tangles. Interestingly, insulin also plays a crucial role in the formation amyloid plaques. In this review, we discussed significant shared mechanisms between AD and diabetes and we also provided therapeutic avenues for diabetes and AD.

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Published in final edited form as:
Biochim Biophys Acta. 2017 May ; 1863(5): 1078–1089. doi:10.1016/j.bbadis.2016.08.018.

Is Alzheimer's disease a Type 3 Diabetes? A Critical Appraisal

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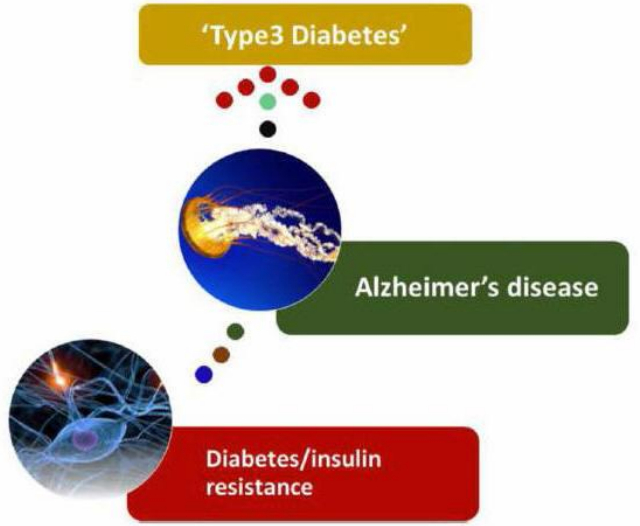

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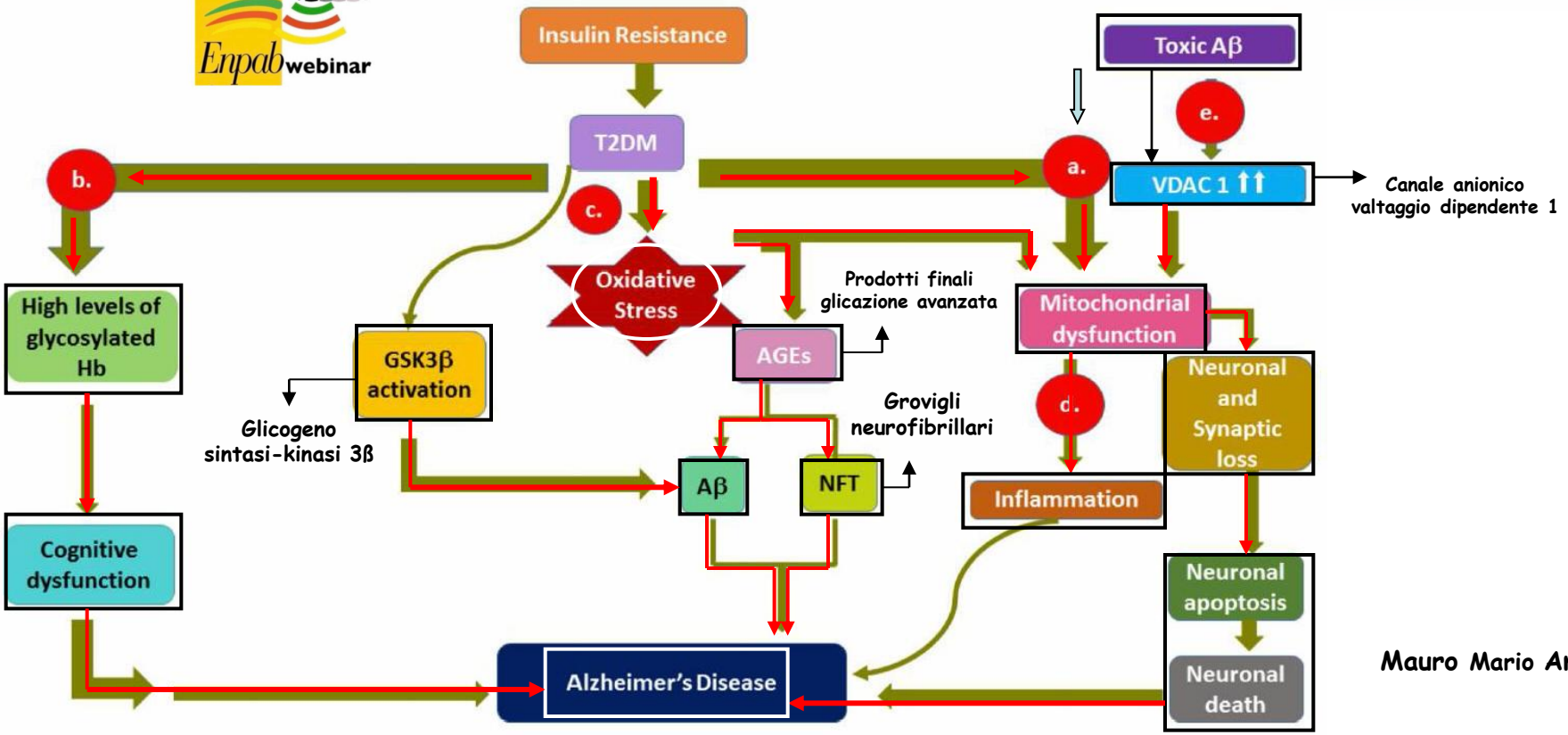

Mauro Mario Amato

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BioMed Research International
Volume 2019, Article ID 1435276, 8 pages
https://doi.org/10.1155/2019/1435276

Review Article

Neurometabolic Evidence Supporting the Hypothesis of Increased Incidence of Type 3 Diabetes Mellitus in the 21st Century

Anna Rorbach-Dolata and Agnieszka Piwowar

Department of Toxicology, Faculty of Pharmacy with the Division of Laboratory Diagnostics, Wroclaw Medical University, Borowska 211, 50-552 Wroclaw, Poland

Correspondence should be addressed to Anna Rorbach-Dolata; anna.rorbach-dolata@umed.wroc.pl

Evidenze neurometaboliche del DMT3

Received 22 February 2019; Accepted 3 July 2019; Published 21 July 2019

Academic Editor: Stavros Baloyannis

ARE YOU AT RISK FOR TYPE 2 DIABETES?

American Diabetes Association.

Diabetes Risk Test

1. **How old are you?**
   Less than 40 years (0 points)
   40—49 years (1 point)
   50—59 years (2 points)
   60 years or older (3 points)

2. **Are you a man or a woman?**
   Man (1 point) Woman (0 points)

3. **If you are a woman, have you ever been diagnosed with gestational diabetes?**
   Yes (1 point) No (0 points)

4. **Do you have a mother, father, sister, or brother with diabetes?**
   Yes (1 point) No (0 points)

5. **Have you ever been diagnosed with high blood pressure?**
   Yes (1 point) No (0 points)

Write your score in the box.

Height	piedi	Weight (lbs.)	libbre
4′ 10″	119-142	143-190	191+
4′ 11″	124-147	148-197	198+
5′ 0″	128-152	153-203	204+
5′ 1″	132-157	158-210	211+
5′ 2″	136-163	164-217	218+
5′ 3″	141-168	169-224	225+
5′ 4″	145-173	174-231	232+
5′ 5″	150-179	180-239	240+
5′ 6″	155-185	186-246	247+
5′ 7″	159-190	191-254	255+
5′ 8″	164-196	197-261	262+
5′ 9″	169-202	203-269	270+
5′ 10″	174-208	209-277	278+
5′ 11″	179-214	215-285	286+
6′ 0″	184-220	221-293	294+
6′ 1″	189-226	227-301	302+
6′ 2″	194-232	233-310	311+

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5 **Have you ever been diagnosed with high blood pressure?**

Yes (1 point) No (0 points)

6 **Are you physically active?**

Yes (0 points) No (1 point)

7 **What is your weight status?** ***(see chart at right)***

Add up your score.

If you scored 5 or higher:

You are at increased risk for having type 2 diabetes. However, only your doctor can tell for sure if you do have type 2 diabetes or prediabetes (a condition that precedes type 2 diabetes in which blood glucose levels are higher than normal). Talk to your doctor to see if additional testing is needed.

Type 2 diabetes is more common in African Americans, Hispanics/Latinos, American Indians, and Asian Americans and Pacific Islanders.

Higher body weights increase diabetes risk for everyone. Asian Americans are at increased diabetes risk at lower body weights than the rest of the general public (about 15 pounds lower).

For more information, visit us at diabetes.org or call 1-800-DIABETES (1-800-342-2383)

piedi → libbre →

6′ 0″	184-220	221-293	294+
6′ 1″	189-226	227-301	302+
6′ 2″	194-232	233-310	311+
6′ 3″	200-239	240-318	319+
6′ 4″	205-245	246-327	328+
	(1 Point)	(2 Points)	(3 Points)
	You weigh less than the amount in the left column (0 points)		

Adapted from Bang et al., Ann Intern Med 151:775-783, 2009.
Original algorithm was validated without gestational diabetes as part of the model.

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Mauro Mario Amato

Metric Conversions.

Metric Conversion > Metric Converter > Length Converter > Meters Conversion > Meters to Feet

https://www.metric-conversions.org/length/meters-to-feet.htm

Meters to Feet

1.70

Feet to Meters (Swap Units)

1.70m= 5ft 6.93in

Format	Feet:Inches
Accuracy	3 significant figures

Note: For a pure decimal result please select 'decimal' from the options above the result.

V Show formula

V Show working

V Show result in exponential format

V More information: Meters

V More information: Feet

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Mauro Mario Amato

Metric Conversions.

Metric Conversion > Metric Converter > Weight Converter > Kilograms Conversion > kg to pounds

https://www.metric-conversions.org/weight/kilograms-to-pounds.htm

Kilograms to Pounds (kg to lbs)

75

pounds to kg (Swap Units)

75kg= 165lb 5.55oz

Format	Pounds:Ounces
Accuracy	3 significant figures

Note: For a pure decimal result please select 'decimal' from the options above the result.

V Show formula

V Show working

V Show result in exponential format

V More information: Kilograms

V More information: Pounds

Enpab webinar

Mauro Mario Amato

Alterata Glicemia a digiuno (IFG)

Mauro Mario Amato

Normale Tolleranza al Glucosio 1h alto (NGT-1h High)

ADA/EASD/ESC
2019

IFG (Impaired Fasting Glycaemia)= **100 – 125 mg/dl**

- Prelievo effettuato dopo 8-10 ore di digiuno.
- Durante 3-4 gg precedenti la determinazione, dieta e attività di consuetudine.

IGT (Impaired Glucose Tolerance)= **141 – 199 mg/dl** (cut point 140 mg/dl)

(**2 ore dopo carico orale di glucosio**)

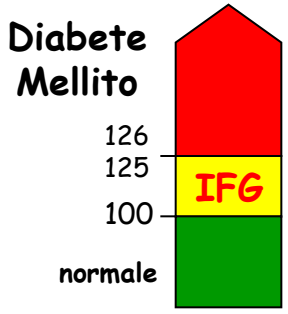
75 gr. di glucosio in soluzione con 200/250 ml di acqua

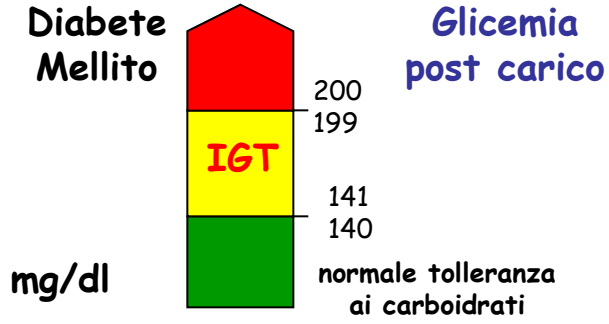
NGT-1h High (Normal Glucose Tolerance – alti alla 1^ora)= **> 155 mg/dl**

(**1 ora dopo carico orale di glucosio**)

75 gr. di glucosio in soluzione con 200/250 ml di acqua

NGT-1h High associato con aumento del rischio per CVD e DMT2
Sebbene **non raccomandato da ADA** per identificazione soggetti con alto rischio diabete, ci sono diverse **evidenze** che pongono questi **soggetti** con un **rischio aumentato di diabete 2** conclamato **entro 5 anni**.

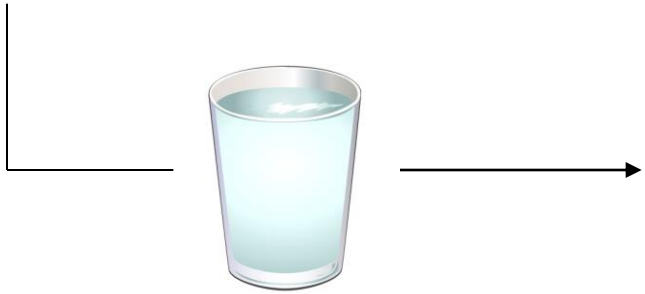




Glicemia base **94 mg/dl**

Sig. Tizio Rossi

75 gr. di glucosio in soluzione con 200/250 ml di acqua



Glicemia 1h **165 mg/dl**
Glicemia 2h **128 mg/dl**

Normale Tolleranza al Glucosio 1h alto
(NGT-1h High)

Cells. 2019 Aug; 8(8): 910.
Published online 2019 Aug 16. doi: 10.3390/cells8080910

PMCID: PMC6721743
PMID: 31426413

1 h Postload Glycemia Is Associated with Low Endogenous Secretory Receptor for Advanced Glycation End Product Levels and Early Markers of Cardiovascular Disease

Antonino Di Pino,† Francesca Urbano,† Roberto Scicali, Stefania Di Mauro, Agnese Filippello, Alessandra Scamporrino, Salvatore Piro, Francesco Purrello,* and Agata Maria Rabuazzo

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NGT-1h high/CVD

Abstract

Go to:

We investigated the correlation of the soluble receptor for advanced glycation end products (sRAGE) and endogenous secretory RAGE (esRAGE) with markers of cardiovascular disease in subjects with normal glucose tolerance (NGT) and 1 h postload glucose ≥155 mg/dL after an oral glucose tolerance test. We stratified 282 subjects without a previous diagnosis of diabetes into three groups: 123 controls (NGT and 1 h postload glycemia <155 mg/dL), 84 NGT and 1 h postload glycemia ≥155 mg/dL (NGT 1 h high), and 75 subjects with impaired fasting glucose and/or impaired glucose tolerance (IFG/IGT). NGT 1 h high subjects exhibited lower esRAGE (0.36 ± 0.18 vs. 0.4 5 ± 0.2, $p < 0.05$) and higher S100A12 levels than controls (5684 (3193.2–8295.6) vs. 3960.1 (2101.8–7419), $p < 0.05$). Furthermore, they showed an increased pulse wave velocity (PWV) and intima–media thickness (IMT). No differences were found

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Endocrine. 2019 Jun;64(3):525-535. doi: 10.1007/s12020-019-01873-5. Epub 2019 Feb 21.

Elevated 1-h post-load plasma glucose is associated with right ventricular morphofunctional parameters in hypertensive patients.

Sciacqua A[1], Perticone M[2], Miceli S[3], Pinto A[3], Cassano V[3], Succurro E[3], Andreozzi F[3], Hribal ML[3], Sesti G[3], Perticone F[3].

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NGT-1h high
Disfunzioni parametri ventricolo dx

Abstract

PURPOSE: Emerging data demonstrate that type 2 diabetes mellitus (T2DM) is associated with right ventricular (RV) dysfunction. A cutoff point of 155 mg/dL for the 1-hour (h) post-load plasma glucose, during oral glucose tolerance test (OGTT), identifies patients with normal glucose tolerance (NGT) at high risk to develop T2DM and cardiovascular (CV) disease. We investigated if 1-h post-load glucose may affect RV geometry and function in a group of never-treated hypertensive individuals.

METHODS: We enrolled 446 Caucasian newly diagnosed hypertensive outpatients. All patients underwent an OGTT and a standard echocardiography. The tricuspid annular plane systolic excursion (TAPSE) and the RV fractional area change (RVFAC) were measured together with systolic pulmonary arterial pressure (s-PAP) and pulmonary vascular resistances (PVR). Insulin sensitivity was evaluated using the Matsuda index.

RESULTS: Among all partecipants, 296 had NGT, 100 impaired glucose tolerance (IGT), and 50 T2DM. Considering the cutoff point of 155 mg/dl for 1-h glucose, NGT subjects were stratified into two groups: NGT < 155 (n = 207), NGT ≥ 155 (n = 89). Subjects NGT ≥ 155 presented a worse metabolic and inflammatory profile than NGT < 155. RV functional parameters (TAPSE, RVFAC, TAPSE/s-PAP, and TAPSE/PVR) were significantly reduced in NGT ≥ 155 subjects compared with NGT < 155 patients. On the contrary, s-PAP and PVR were significantly higher. At multiple regression analysis, 1-h glucose was the strongest predictor of TAPSE in NGT ≥ 155, IGT, and T2DM.

CONCLUSIONS: The presence of RV impairment in hypertensive NGT ≥ 155 subjects further complicates their CV burden and it may, at least in part, justify the worse clinical outcome in this setting of patients.

https://www.ncbi.nlm.nih.gov/pubmed/30790176

Mauro Mario Amato

atherosclerosis

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< Previous Article · January 2017 Volume 256, Pages 15–20 · Next Article >

Elevated 1-h post-load plasma glucose levels in subjects with normal glucose tolerance are associated with a pro-atherogenic lipid profile

Francesco Andreozzi, Gaia C. Mannino, Maria Perticone, Francesco Perticone, Giorgio Sesti*

Department of Medical and Surgical Sciences, University Magna Graecia of Catanzaro, Catanzaro, Italy

PlumX Metrics

DOI: https://doi.org/10.1016/j.atherosclerosis.2016.11.020 | Check for updates

Article Info

Abstract · Full Text · References

Highlights

- Diabetes and pre-diabetes at diagnosis already possess atherogenic dyslipidemia.
- Fasting, 1-h and 2-h glycemia strongly modulate individual's lipid pattern.
- NGT-1h-high ≥155 mg/dl have a worse cardio-metabolic profile than plain NGT.

NGT-1h high/Profilo lipidico aterogenico

https://www.atherosclerosis-journal.com/article/S0021-9150(16)31488-5/fulltext

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https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5319479/

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PMCID: PMC5319479
NIHMSID: NIHMS841486
PMID: 26419850

Elevated 1-Hour Plasma Glucose Levels are Associated With Dysglycemia, Impaired Beta-Cell Function and Insulin Sensitivity: Pilot Study From a Real World Health Care Setting

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Introduction

Go to:

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Associazione
NGT-1h high/Disfunzioni glicemiche
NGT-1h high/Disfunzioni ß-cellule
NGT-1h high/Sensibilità insulinica

Mauro Mario Amato

Diabetologia. 2019; 62(3): 544–548.
Published online 2018 Dec 29. doi: 10.1007/s00125-018-4798-5

PMCID: PMC6428784
PMID: 30594956

One hour post-load plasma glucose and 3 year risk of worsening fasting and 2 hour glucose tolerance in the RISC cohort

Melania Manco,[✉1] Andrea Mari,[2] John Petrie,[✉3] Geltrude Mingrone,[4,5] Beverley Balkau,[6] and for the EGIR-RISC study group

Author information ▸ Article notes ▸ Copyright and License information Disclaimer

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Associazione
NGT-1h high/rischio diabete a 3 anni

Mauro Mario Amato

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6428784/

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J Clin Endocrinol Metab. 2018 Sep 1;103(9):3131-3143. doi: 10.1210/jc.2018-00468.

One-Hour Postload Hyperglycemia: Implications for Prediction and Prevention of Type 2 Diabetes.

Fiorentino TV[1], Marini MA[2], Succurro E[1], Andreozzi F[1], Perticone M[3], Hribal ML[1], Sciacqua A[1], Perticone F[1], Sesti G[1].

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3 Department of Experimental and Clinical Medicine, University Magna Græcia of Catanzaro, Viale Europa, Catanzaro, Italy.

Abstract

CONTEXT: Recently, a value of 1-hour postload glucose concentration (1-h-PG) ≥155 mg/dL (8.6 mmol/L) in individuals with normal glucose tolerance (NGT) has been found to be associated with an increased risk for future type 2 diabetes mellitus (T2DM). In this review, we analyze the implication of 1-h-PG determination in prediction of T2DM and cardiovascular disease.

DESIGN: A literature search was performed using MEDLINE. We included all English studies published up to February 2018 in peer-reviewed journals that examined the relationship between 1-h-PG and diabetes, cardiometabolic alterations, organ damage, and cardiovascular disease.

RESULTS: Several longitudinal studies have consistently shown that 1-h-PG ≥155 mg/dL can recognize individuals at increased risk for future T2DM among subjects with NGT. Additionally, we describe the pathophysiological abnormalities associated with 1-h-PG ≥155 mg/dL including impaired insulin sensitivity, β-cell dysfunction, and increased glucose intestinal absorption, which are known to be involved in T2DM pathogenesis. Importantly, numerous studies have demonstrated that a value of 1-h-PG ≥155 mg/dL in individuals with NGT is not only linked to an increased risk for future T2DM, but also able to identify those having a worse cardiovascular phenotype and an increased risk of adverse cardiovascular outcomes.

CONCLUSIONS: Although 1-h-PG determination is not currently recommended by the American Diabetes Association for identifying high-risk individuals, the available evidence indicates that a value of 1-h-PG ≥155 mg/dL may be a useful tool to recognize, among subjects with NGT, those at increased risk of T2DM and cardiovascular disease.

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NGT-1h high indicatore per prevedere/prevenire DMT2

https://www.ncbi.nlm.nih.gov/pubmed/30020454/

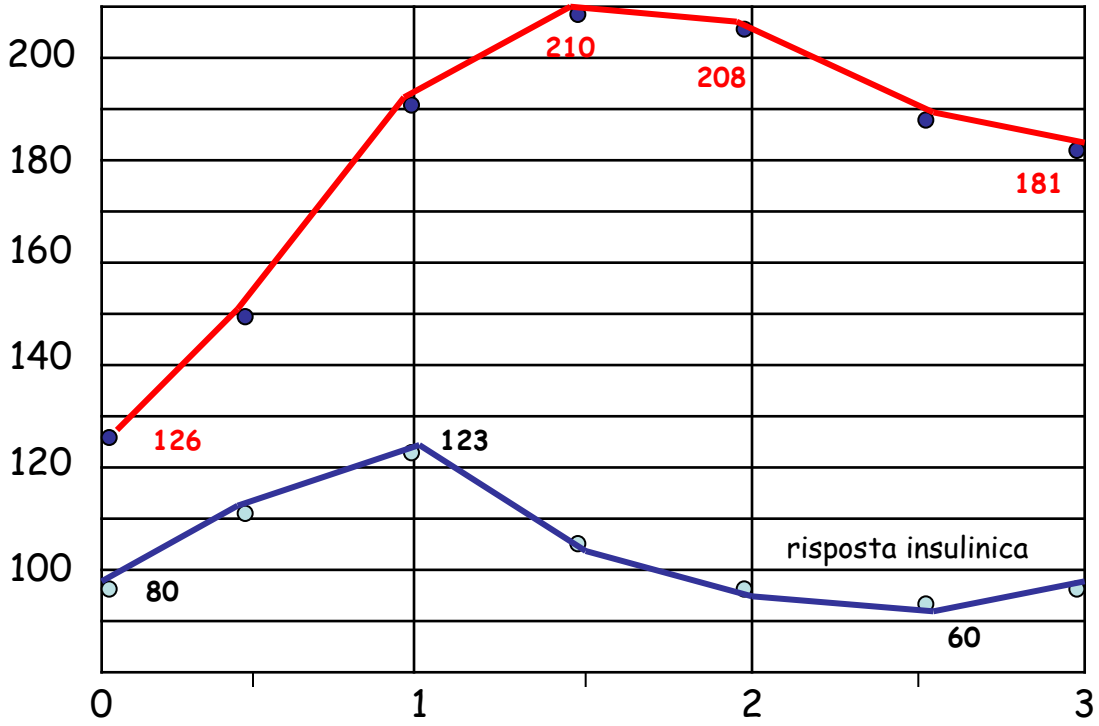
Mauro Mario Amato

Curva Glicemica da Carico orale di Glucosio (OGTT)

Consente la **conferma** della **condizione di DMT2** soprattutto quando i dati clinico-diagnostici non danno risposte sufficienti.

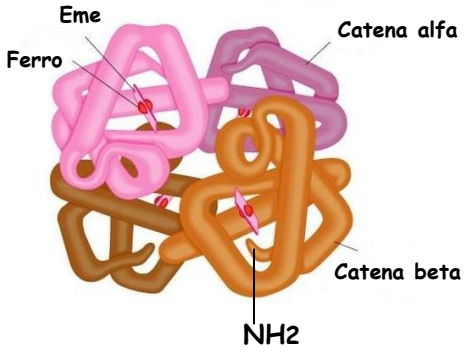
OGTT (Oral Glucose Tolerance Test)

- Determinazione glicemia a digiuno (**glicemia base**).
- Somministrazione orale di **75 gr** di glucosio in soluzione con 200/250 ml di acqua (in caso soggetti con **peso inferiore a 45 kg** la quantità sarà di **1.75 gr./kg corporeo**); tale soluzione bevuta con tranquillità entro 2/4 minuti.
- Prelievi ematici eseguiti a **30', 60', 90', 120' con determinazione glicemica**.
- I **tempi possono variare**, a seconda delle necessità individuali rilevate dal clinico, **da un solo prelievo dopo 2 ore**, a **prelievi seriati** che possono arrivare anche **a 180'**.

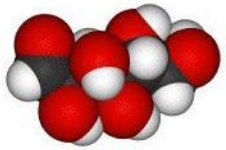


Le curve riportate sono **andamenti tipici**. Non ci sono ben precisi valori di riferimento per l'OGTT in quanto la risposta al carico può variare in relazione a diversi **fattori individuali** (come età, sesso, condizioni fisio-patologiche) a conoscenza del **clinico che valuterà l'andamento generale della curva stessa**

EMOGLOBINA GLICATA (HbA1c) (Dosaggio Immunoturbidimentrico)



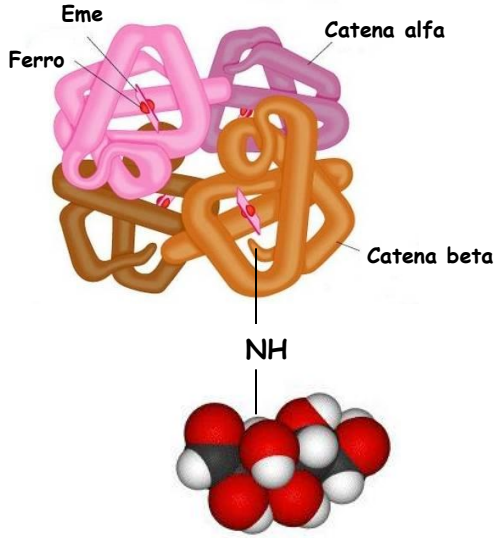
Emoglobina A1



Glucosio

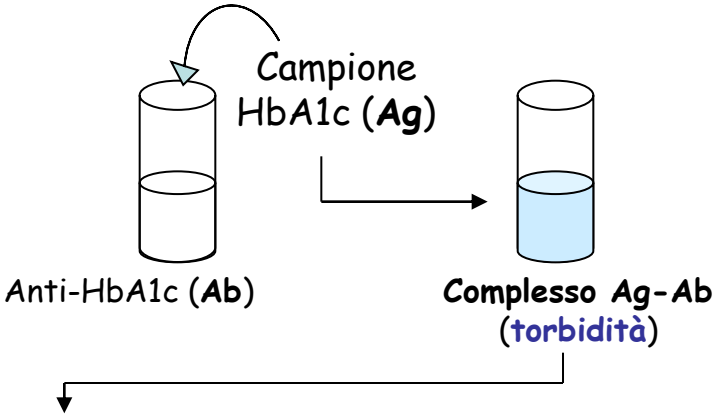
esposizione prolungata della emoglobina A a concentrazioni d glucosio più o meno elevate

glicazione



Emoglobina glicata (HbA1c)

- **Indicatore andamento glicemico nel periodo di 2-3 mesi precedenti** il dosaggio della glicemia essendo la vita media dei GR di circa 120 gg.
- Si ritiene che la **percentuale più cospicua** sia da associare all'esposizione al glucosio avvenuto **nelle 3-4 settimane precedenti** la determinazione.



dovuta alla formazione degli immunocomplessi (Ag-Ab) la cui intesità, letta da uno strumento opportuno, è funzione della quantità di HbA1c presente nel campione in esame.

Dosaggio immunoturbidimetrico

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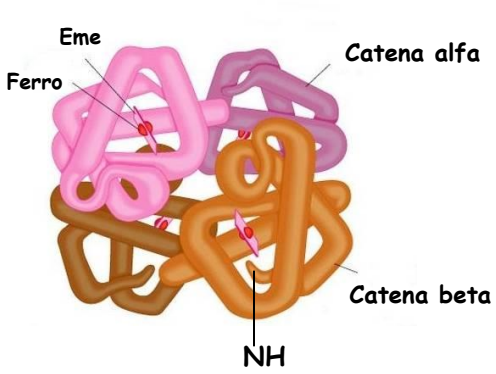
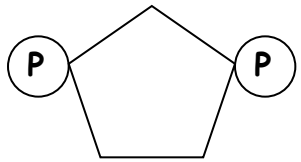
EMOGLOBINA A1 / EMOGLOBINE Glicate



Emoglobine A1 glicate (HbA1) → N-glicazione con il gruppo NH2 terminale della catena beta dell'emoglobina.
(condizioni normali 5-7%)

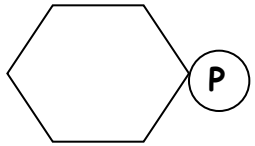
Glicazione

Emoglobina A1 (HbA1)
(condizioni normali 93-95%)



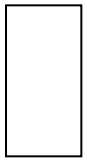
fruttosio 1,6 difosfato

HbA1a1 (0.25%)



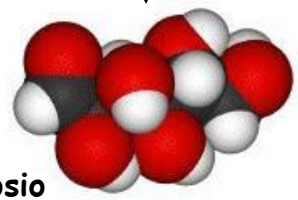
Glucosio 6 fosfato

HbA1a2 (0.25%)



Piruvato

HbA1b (0.5%)



Glucosio

HbA1c (4-6%)

- Componente maggiore di emoglobina glicata -
- **Glicazione molto affine** con il glucosio

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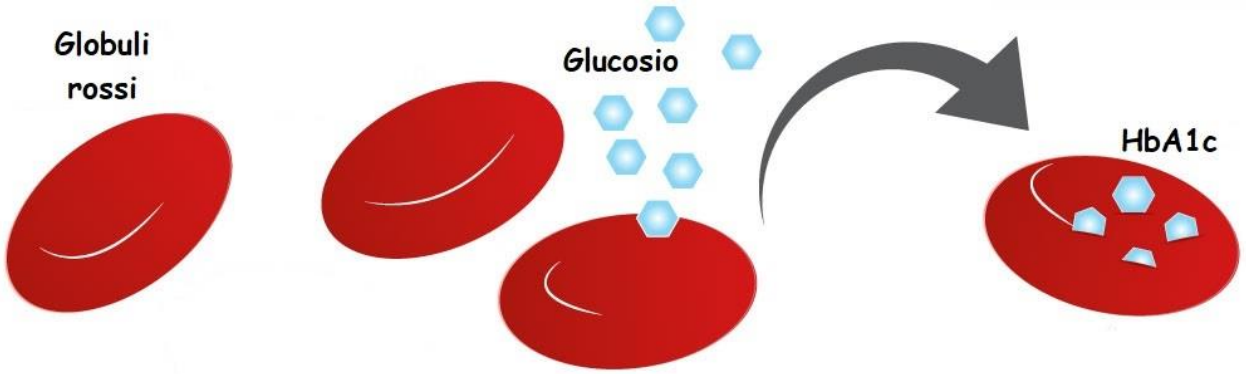
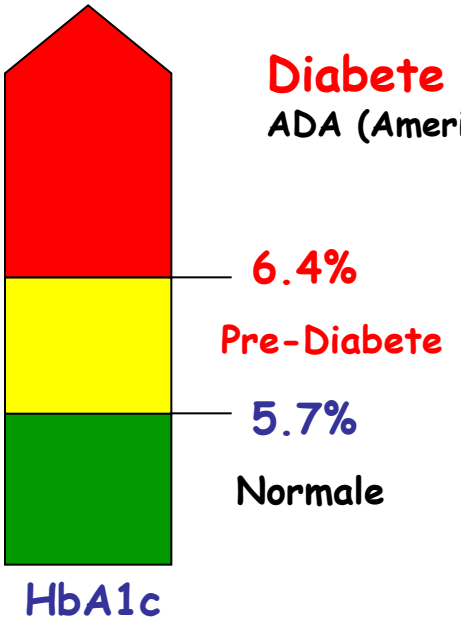
EMOGLOBINA GLICATA (HbA1c)



Livelli

HbA1c %	Glicemia media (mg/dl)
5	90
6	120
7	150
8	180
9	210
10	240
11	270
12	300



Diabete linee guida:
ADA (American Diabetes Association) 2019

Mauro Mario Amato

Table 2.5—Criteria defining prediabetes*
FPG 100 mg/dL (5.6 mmol/L) to 125 mg/dL (6.9 mmol/L) (IFG)
OR
2-h PG during 75-g OGTT 140 mg/dL (7.8 mmol/L) to 199 mg/dL (11.0 mmol/L) (IGT)
OR
A1C 5.7–6.4% (39–47 mmol/mol)

*For all three tests, risk is continuous, extending below the lower limit of the range and becoming disproportionately greater at the higher end of the range.

ADA Diabetes 2019

Table 2.2—Criteria for the diagnosis of diabetes
FPG ≥126 mg/dL (7.0 mmol/L). Fasting is defined as no caloric intake for at least 8 h.*
OR
2-h PG ≥200 mg/dL (11.1 mmol/L) during OGTT. The test should be performed as described by the WHO, using a glucose load containing the equivalent of 75-g anhydrous glucose dissolved in water.*
OR
A1C ≥6.5% (48 mmol/mol). The test should be performed in a laboratory using a method that is NGSP certified and standardized to the DCCT assay.*
OR
In a patient with classic symptoms of hyperglycemia or hyperglycemic crisis, a random plasma glucose ≥200 mg/dL (11.1 mmol/L).

*In the absence of unequivocal hyperglycemia, diagnosis requires two abnormal test results from the same sample or in two separate test samples.

ADA Diabetes 2019

Linee Guida
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Table 19.2.2 Cut-points for diagnosing diabetes, impaired glucose tolerance, and impaired fasting glucose based on other blood specimens than the recommended standard, venous plasma

Diagnosis	Venous plasma[a] mmol/L (mg/dL)	Venous whole blood mmol/L (mg/dL)	Capillary blood mmol/L (mg/dL)
IFG: FG	6.1 (110)	5.0 (90)	5.6 (101)
IGT: 2hG	7.8 (140)	6.5 (117)	7.2 (130)
Diabetes: FG	7.0 (126)	5.8 (104)	6.5 (117)
Diabetes: 2hG	11.1 (200)	9.4 (169)	10.3 (185)

[a] Standard.

2hG, 2 h post-load glucose; 2hPG, 2 h post-load plasma glucose; FPG, fasting plasma glucose; FG, fasting glucose; IFG, impaired fasting glucose; IGT, impaired glucose tolerance.

Published on behalf of European Society of Cardiology

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Use of Glycated Haemoglobin (HbA1c) in the Diagnosis of Diabetes Mellitus

Abbreviated Report of a WHO Consultation

World Health Organization

Enpab webinar

Materiale Didattico

Mauro Mario Amato

SUPPLEMENT 1

AMERICAN DIABETES ASSOCIATION

STANDARDS OF MEDICAL CARE IN DIABETES—2019

Materiale Didattico

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Da qualche tempo è stata introdotta una **nuova unità di misura per l'HbA1c** dall'IFCC (International Federation of Clinical Chemistry and Laboratory Medicine) corrispondente alle **mmoli/mole** di emoglobina che andrà a sostituire il valore % definito dalla NGSP. Sotto la tabella di conversione:

HbA1c %	HbA1c mmoli/mole	HbA1c %	HbA1c mmoli/mole
4.0	20	8.1	65
4.1	21	8.2	66
4.2	22	8.3	67
4.3	23	8.4	68
4.4	25	8.5	69
4.5	26	8.6	70
4.6	27	8.7	72
4.7	28	8.8	73
4.8	29	8.9	74
4.9	30	9.0	75
5.0	31	9.1	76
5.1	32	9.2	77
5.2	33	9.3	78
5.3	34	9.4	79
5.4	36	9.5	80
5.5	37	9.6	81
5.6	38	9.7	83
5.7	39	9.8	84
5.8	40	9.9	85
5.9	41	10	86
6.0	42	10.1	87
6.1	43	10.2	88
6.2	44	10.3	89
6.3	45	10.4	90
6.4	46	10.5	91
6.5	48	10.6	92
6.6	49	10.7	93
6.7	50	10.8	95
6.8	51	10.9	96
6.9	52	11.0	97
7.0	53	11.1	98
7.1	54	11.2	99
7.2	55	11.3	100
7.3	56	11.4	101
7.4	57	11.5	102
7.5	58	11.6	103
7.6	60	11.7	104
7.7	61	11.8	105
7.8	62	11.9	107
7.9	63	12.0	108
8.0	64		

Mauro Mario Amato

Convertitore online per HbA1c → http://glyconverter.altervista.org/

Fornisce anche il valore della glicemia media

DIABETE MELLITO Tipo 1 (T1DM)

Cause principali:
- Distruzione autoimmune ß-cellule
- Danni/Rimozione Pancreas
- Malattie endocrine o autoimmuni
- Infezioni virali/batteriche
- Predisposizione ereditaria
- Farmaci/Tossine chimiche

Sintomi:
- Assetato con frequente minzione
- Bocca secca con nausea e vomito
- Dolori addominali e perdita di peso
- Debole e sempre affamato
- Sindrome del piede diabetico
- Disturbi visivi

Autoimmunità

- **Anti-Insula pancreatica** (anti-ICA)
- **Anti-Decarbossilasi Acido Glutammico** (anti-GAD65)
- **Anti-Tirosina Fosfatasi insulare** (anti-IA2)
- **Anti-Trasportatore-Zn 8** (anti-ZnT8)

*"Type 1 diabetes is defined by the **presence of one or more** of these autoimmune markers"*
(ADA 2019)

- **Anti-Insulina** (anti-IAA)

laboratorio

- **Glicemia a digiuno** >125 mg/dl
- **Glicemia random** >= 200 mg/dl
- **HbA1c** > 6.5% (48 mmoli/mole)
- **Insulinemia**

Identificazione soggetti a rischio

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DIABETE MELLITO Tipo 1 (T1DM)

TYPE 1 DIABETES

Recommendations

2.4 Plasma blood glucose rather than A1C should be used to diagnose the acute onset of type 1 diabetes in individuals with symptoms of hyperglycemia. **E**

2.5 Screening for type 1 diabetes risk with a panel of autoantibodies is currently recommended only in the setting of a research trial or in first-degree family members of a proband with type 1 diabetes. **B**

2.6 Persistence of two or more autoantibodies predicts clinical diabetes and may serve as an indication for intervention in the setting of a clinical trial. **B**

ADA Diabetes 2019

Soggetti T1DM1 inclini ad **altri disturbi autoimmuni**:

- Tiroidite di Hashimoto
- Morbo di Graves
- Celiachia
- Vitiligine
- Epatite autoimmune
- Miastenia grave

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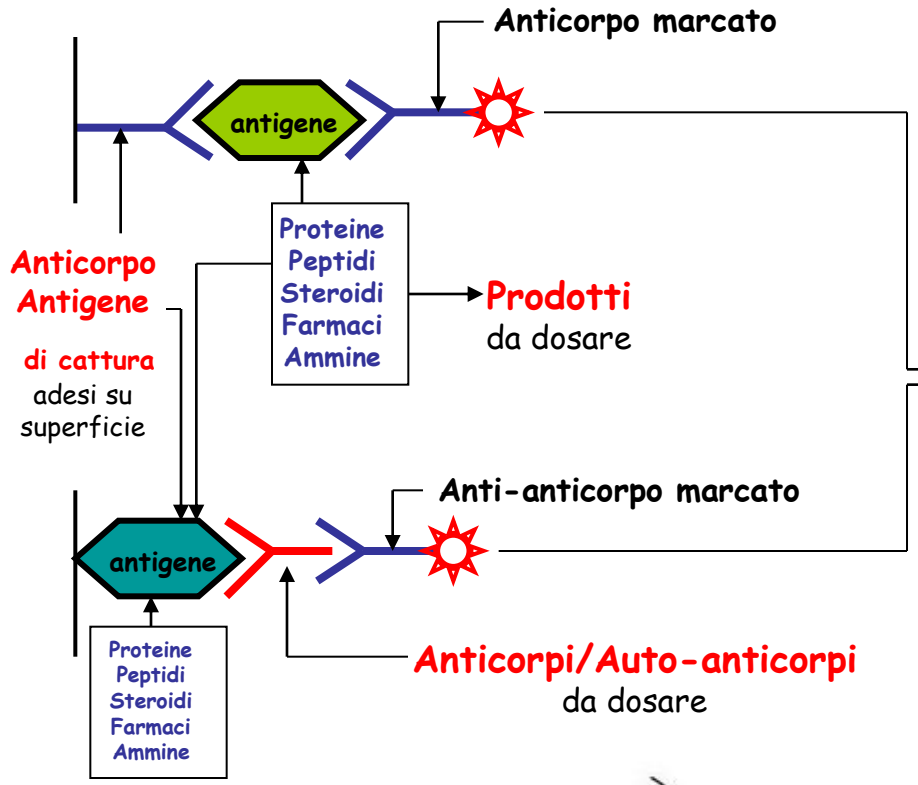
Schema di base del
Principio di Azione
Tecniche Immunochimiche

Mauro Mario Amato



Isotopi (RIA, IRMA)
Sostanze fluorescenti (FIA, FPIA, MEIA)
Sostanze luminescenti (CLIA, LIA)
Enzimi (ELISA)

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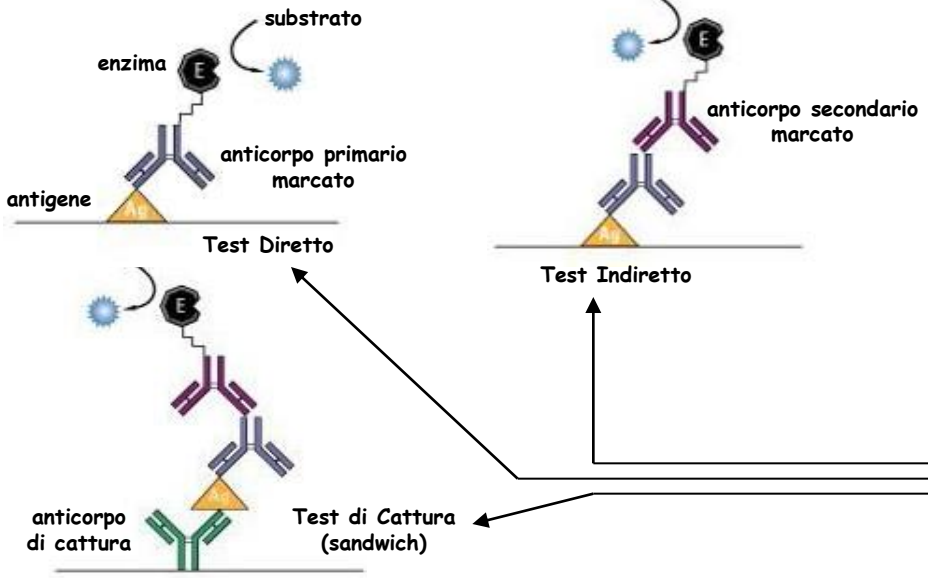


Legenda
RIA= Radio Immuno Assay
IRMA= Immunoradiometric Assay
FIA= Fluorescent Immuno Assay
FPIA= Fluorescent Polarized Immuno Assay
MEIA= Microparticle Enzyme Immuno Assay
CLIA= Chemi Luminescent Immuno Assay
LIA= Luminescent Immuno Assay
ELISA= Enzyme-Linked Immunosorbent Assay

ELISA

INSULINA

Ormone proteico (cellule beta pancreas endocrino) **ipoglicemizzante** e **anabolico** per eccellenza: interviene anche su metabolismo lipidico e protidico:

- **Stimola trasporto del glucosio** in tutti i tessuti, particolarmente nelle **cellule adipose e muscolari**.
- **Attiva** la captazione del glucosio a livello epatico e muscolare per la **sintesi del glicogeno**.
- **Diminuisce** la **formazione di glucosio intracellulare** (soprattutto **gluconeogenesi epatica**)
- **Riduce** la **degradazione dei trigliceridi** nel tessuto adiposo favorendone il deposito.
- **Azione lipogenetica** **tessuto adiposo** (carboidrati → AcetilCoA → Ac. palmitico)
- **Inibisce** liberazione acidi grassi e **chetogenesi**.
- **Inibisce** il **catabolismo proteico**
- **Regola** la **sensazione di fame** a livello ipotalamico

La cellula beta è anche sensore
secrezione insulinica regolata feed back dalla glicemia:
- **valori =< a 50 mg/dl è quasi nulla**
- **valori => a 250 mg/dl è massima**

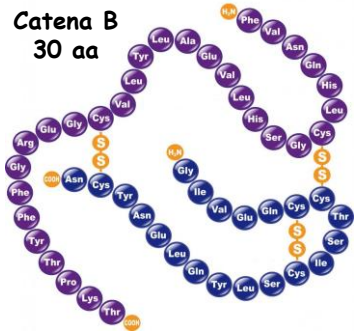
Livelli aumentati:
- Resistenza insulinica
- Diabete tipo 2
- Diabete di natura endocrina
- Obesità
- Intolleranza fruttosio/galattosio
- Acromegalia e Sindrome di Cushing
- Insulinoma
- Farmaci: corticosteroidi, levodopa, contraccettivi orali

Livelli diminuiti:
- Diabete tipo 1
- Diabete tipo 2 in fase avanzata
- Diabete post-pancreatite
- Ipopituitarismo

Dosaggio Immunochimico

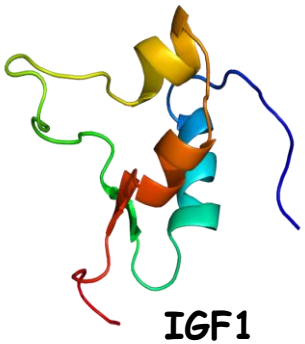
valori di riferimento

4 - 23 µU/ml

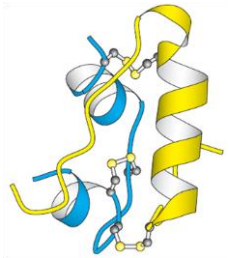


Insulina Umana

IGF1 (Insulin-like Growth Factor 1)
Somatomedina C



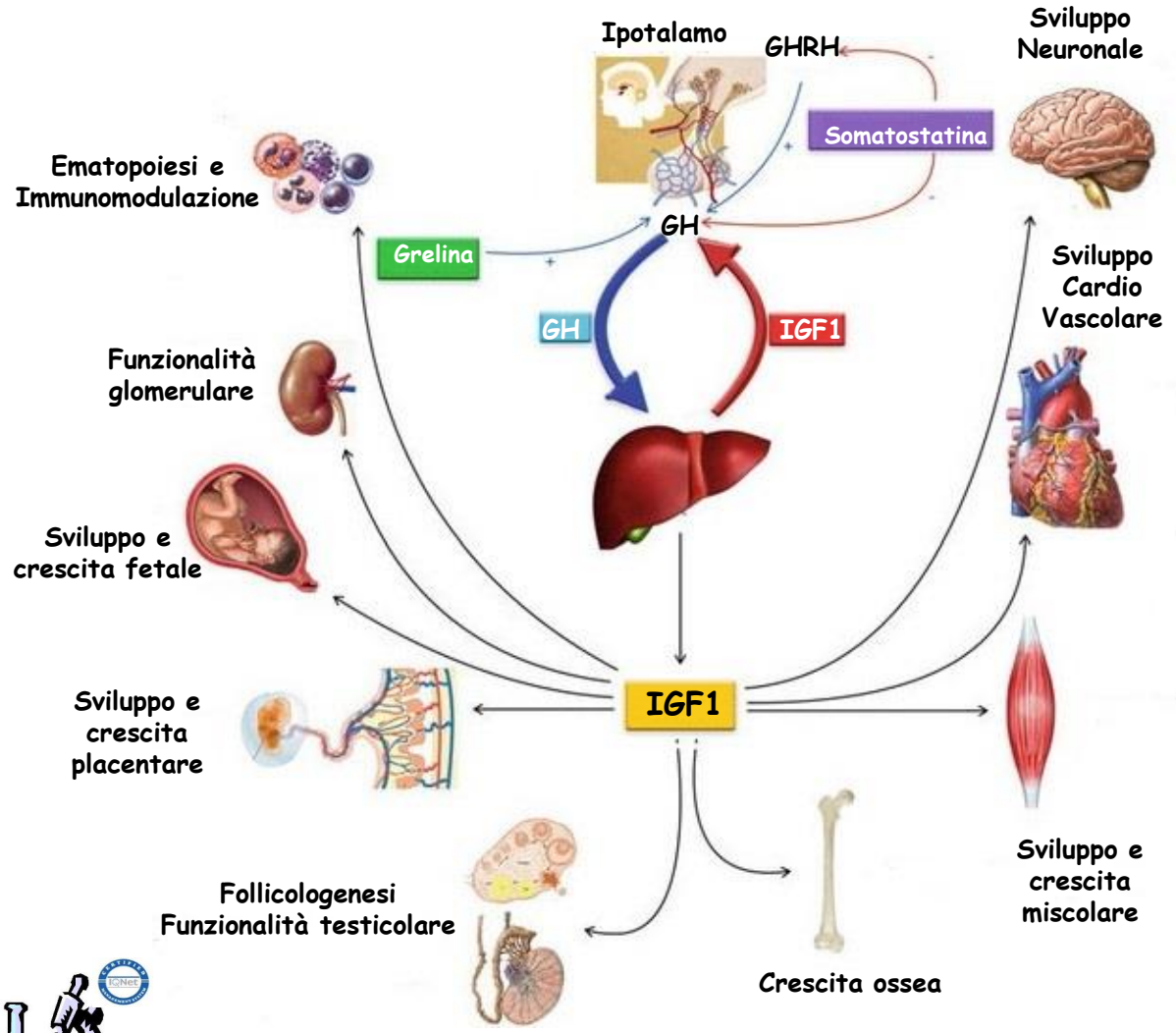
IGF1



Insulina

Ormone simile nella struttura molecolare all'insulina
Ruolo **importante nella crescita** con **effetti anabolici negli adulti**

Prodotto principalmente dal fegato sotto stimolo del GH
La maggior parte dell'**IGF-1** è **legata alla IGF-BP3** (proteina legante IGF1) a sua volta **regolata dall'insulina**
Prodotto per tutta la vita con i livelli più bassi nell'infanzia e nella vecchiaia e più alti in puberta

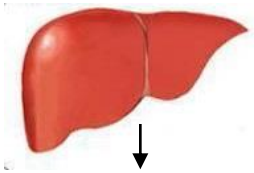


Growth Hormone (GH)

Human Growth Hormone (HGH)

Somatotropo (STH)

(emivita 5-6 min.)



IGF-1 (Somatomedina C)

(emivita 15-18h)

Livelli plasmatici più stabili

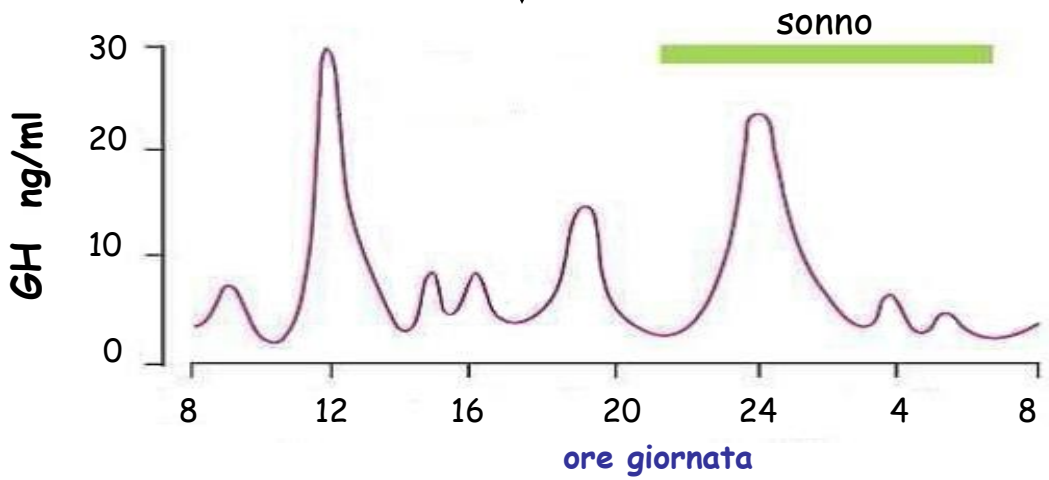
Dosaggio immunochimico

5- 7 anni	**50-310**
8- 10 anni	**65-450**
11-13 anni	**100-750**
14-18 anni	**160-850**
19-25 anni	**115-450**
26-36 anni	**115-300**
37-60 anni	**80-250**

regolati da:

- Stato nutrizionale
- Fattori genetici
- IGF-BP
- Ormoni sessuali
- **Insulina**
- Cortisolo
- Tiroxina
- Età

Livelli plasmatici pulsanti



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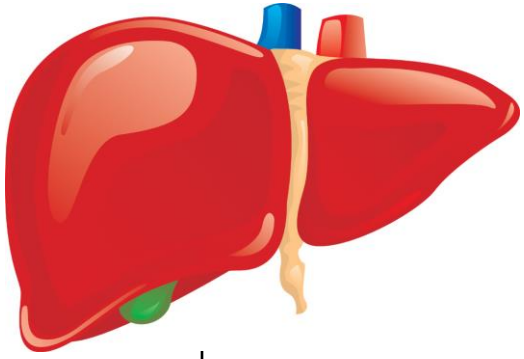
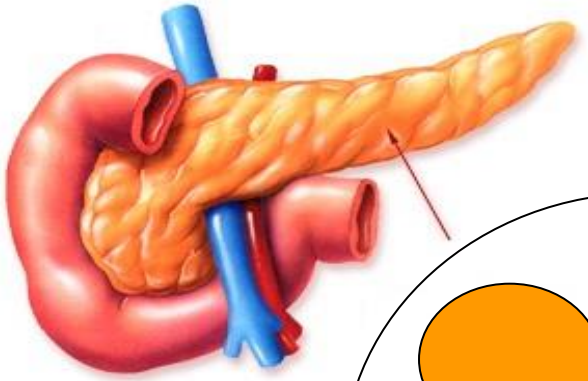
INSULINA (Feed Back negativo)



Insulina ↑

Bassa affinità



Rilascio di **Insulina** ↓

AMP ciclico ↓

PDE3B ↑
Fosfodiesterasi 3B

IGF-1 ↑
Somatomedina C

Alta affinità

IGF-1
recettore

Cellula β Pancreatica

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RESISTENZA INSULINICA (Insulino-resistenza)

Situazione in cui una certa dose di insulina evoca una risposta biologica anormale

Meccanismi

Anomalie del prodotto insulinico
- Molecola anomala.
- Conversione incompleta della pro-insulina.

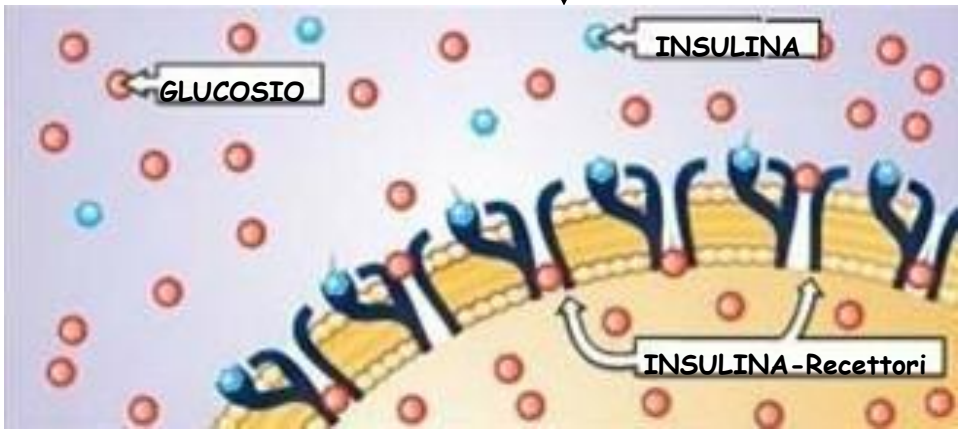
Presenza antagonisti insulinici in circolo
- Elevate concentrazioni di ormoni antagonisti.
- Presenza di anti-insulina e/o anti-insula pancreatica.

Anomalie dei siti bersaglio
- Diminuzione numero recettori cellulari.
- Difetto post-recettoriale.

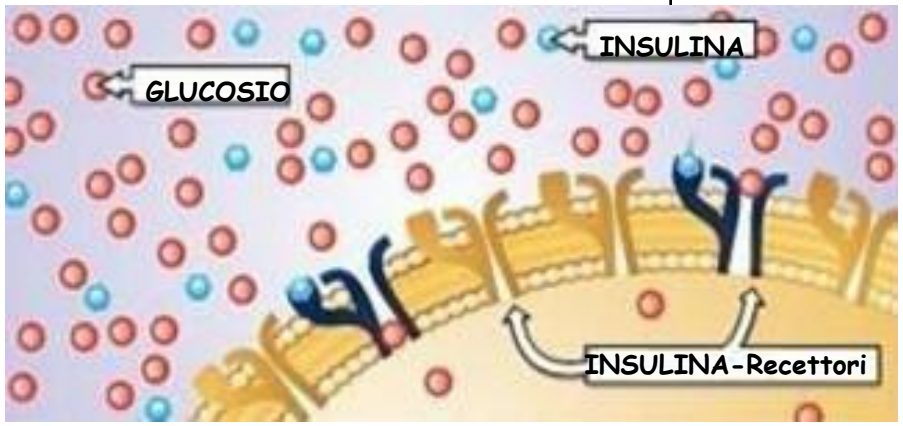
L'insulina deve reagire con i **recettori specifici di membrana** per svolgere la sua **attività ipoglicemizzante**.

L'**affinità recettori-insulina** si abbassa e l'organismo compensa stimolando il pancreas a produrre più insulina (**iperinsulinemia**).

CELLULA NORMALE



CELLULA INSULINO-RESISTENTE



Eccesso di peso
Assunzione eccessiva di carboidrati
Stile di vita sedentaria
Predisposizione genetica

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Aumento grasso intra-addominale

RESISTENZA INSULINICA

Gonfiore
Vertigini
Stitichezza
Bruciore di stomaco
Disfunzione erettile

Aumento della Glicemia

Infiammazione epatica

Ipertensione

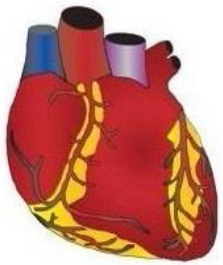
Aterosclerosi

Cardiomiopatia

Apnea del sonno

Rischio di trombosi

PCOS



Diminuzione HDL
Aumento Trigliceridi
LDL small dense

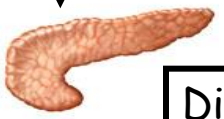
Mauro Mario Amato

Obesità
(adiposità viscerale)

Dieta iperlipidica	Farmaci
Scarsa attività fisica	Componente genetica

Fumo di sigararetta

INSULINO-RESISTENZA



Diabete Mellito 2

Squilibrio ormonale

Ovaio policistico

PCOS

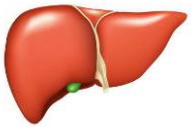
Ipotiroidismo

Tessuto adiposo ↑

Colesterolo ↑
Trigliceridi ↑
HDL ↓

Sintesi acidi grassi

Insulina ↑



Inibizione **SHBG**

Ormoni Androgeni ↑

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Insulin Resistance

Andrew M. Freeman; Kristina Soman-Faulkner; Nicholas Pennings.

Author Information

Authors

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[2] Campbell University

Last Update: May 10, 2019.

https://www.ncbi.nlm.nih.gov/books/NBK507839/

Introduction

Go to:

Insulin resistance is identified as an impaired biologic response to insulin stimulation of target tissues, primarily liver, muscle, and adipose tissue. Insulin resistance impairs glucose disposal, resulting in a compensatory increase in beta-cell insulin production and hyperinsulinemia. [1][2][3]The metabolic consequences of insulin resistance can result in hyperglycemia, hypertension, dyslipidemia, visceral adiposity, hyperuricemia, elevated inflammatory markers, endothelial dysfunction, and a prothrombic state. Progression of insulin resistance can lead to metabolic syndrome, nonalcoholic fatty liver disease (NAFLD), and type 2 diabetes mellitus.

Insulin resistance is primarily an acquired condition related to excess body fat, though genetic causes are identified as well. The clinical definition of insulin resistance remains elusive as there is not a generally accepted test for insulin resistance. [4][5]Clinically, insulin resistance is recognized via the metabolic consequences associated with insulin resistance as described in metabolic syndrome and insulin resistance syndrome.

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Mauro Mario Amato

Molecular Mechanisms of Insulin Resistance: An Update

Citlaly Gutiérrez-Rodelo, Adriana Roura-Guiberna and Jesús Alberto Olivares-Reyes

Laboratory of Signal Transduction, Department of Biochemistry, Centro de Investigación y de Estudios Avanzados del Instituto Politécnico Nacional (IPN), Mexico City, Mexico

Aggiornamenti sui meccanismi molecolari insulino-resistenza

Abstract

The biological actions of insulin are initiated by activating its membrane receptor, which triggers multiple signaling pathways to mediate their biological actions. Due to the importance of metabolic regulation and promoting functions of cell growth and proliferation, insulin actions are highly regulated to promote proper metabolic functioning and energy balance. If these mechanisms are altered, this can lead to a condition known as insulin resistance, which is the consequence of a deficient insulin signaling caused by mutations or post-translational modifications of the receptor or effector molecules located downstream. Insulin resistance is one of the main characteristics of pathological manifestations associated with type 2 diabetes mellitus, one of the leading causes of death in Mexico and worldwide. In recent years, it has been found that conditions such as inflammation, endoplasmic reticulum stress, and mitochondrial dysfunction promote insulin resistance. The aim of this review is to elucidate the molecular aspects of insulin resistance and the mechanisms involved in regulating its effects, with particular emphasis on the role of inflammation, endoplasmic reticulum stress, and mitochondrial dysfunction.

KEY WORDS: *Insulin. Insulin resistance. Inflammation. Endoplasmic reticulum stress. Mitochondrial dysfunction.*

https://www.researchgate.net/publication/317837532_Molecular_Mechanisms_of_Insulin_Resistance_An_Update

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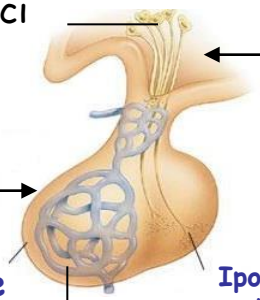
Cellule neurosecretrici

Ipotalamo

GnRH ↑



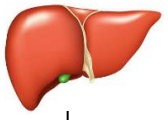
Ipofisi anteriore
Adenoipofisi

Ipofisi posteriore
Neuroipofisi

- Predisposizione Genetica
- Obesità
- Stile di vita sedentaria
- Esposizione prenatale agli androgeni (?)

Resistenza Insulinica

Iperinsulinemia



SHBG ↓

LH ↑ FSH

Rapporto LH/FSH ↑

Ovaio

Cellule Teca follicolare

- Irsutismo
- Acne
- Alopecia

Eccesso di Androgeni

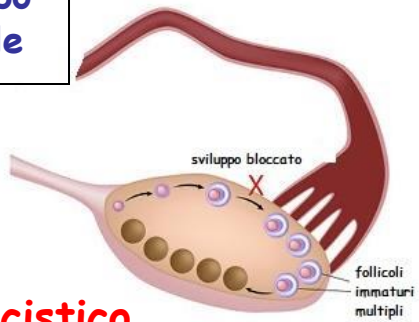
Arresto sviluppo follicolo antrale

- Diabete Mellito Tipo 2
- Dislipidemia
- Problemi cardiovascolari

no corpo luteo

Progesterone ↓

Anovulazione



Ovaio policistico

Rapporto Estrogeni/Progesterone ↑

Iperplasia Endometrio

Neoplasia Endometrio

PCOS

Expert Rev Endocrinol Metab. Author manuscript; available in PMC 2020 Jan 30.

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PMCID: PMC6992448
NIHMSID: NIHMS1067323
PMID: 30767580

Hyperandrogenic Origins of Polycystic Ovary Syndrome – Implications for Pathophysiology and Therapy

David H Abbott,[1,3] Daniel A Dumesic,[4] and Jon E Levine[1,2]

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https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6992448/

Abstract

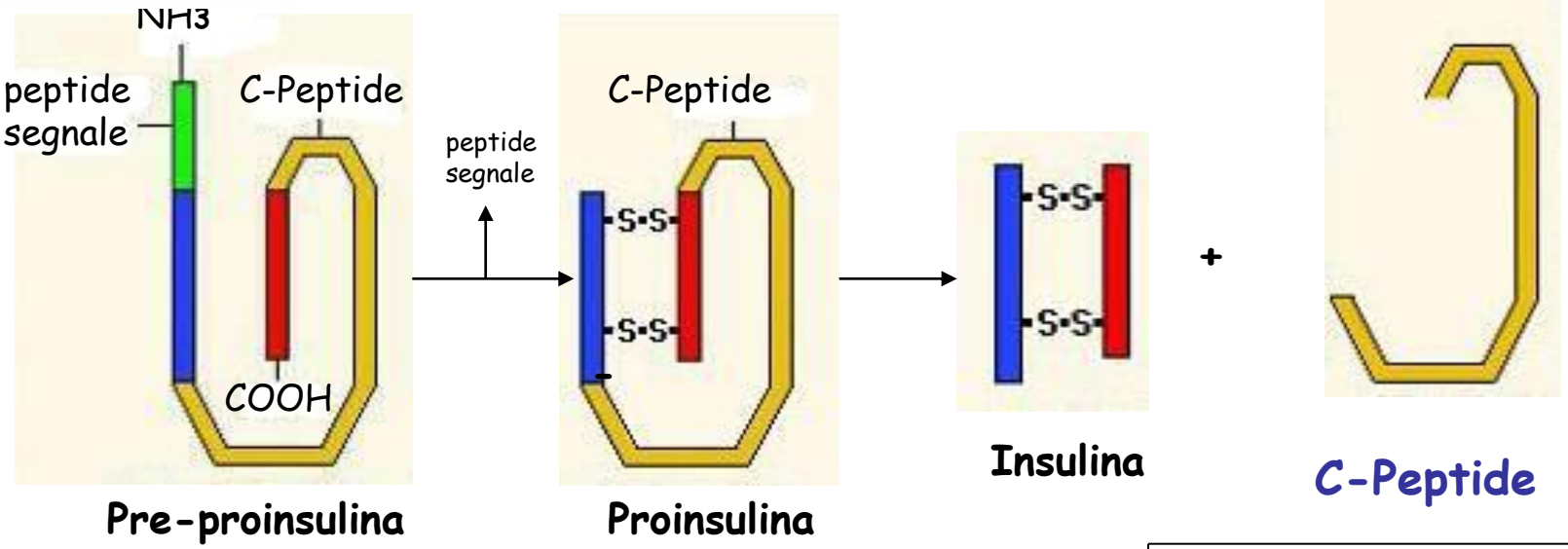
Introduction:

Polycystic ovary syndrome (PCOS) diagnosis comprises combinations of female hyperandrogenism, menstrual irregularity and polycystic ovaries. While it is a familial and highly prevalent endocrine disorder, progress towards a cure is hindered by absence of a definitive pathogenic mechanism and lack of an animal model of naturally occurring PCOS.

Origine Iperandrogenica del PCOS
Esposizione prenatale

Mauro Mario Amato

METABOLISMO GLUCIDICO: C-Peptide



Prodotto **equimolare** con insulina,

- **Emivita più lunga** (25 min. sui 4 dell'insulina) per cui valori più stabili
- **Metabolizzato a livello renale** con clearance costante (dosato nelle urine)
- **Non reagisce** con gli anticorpi antinsulina

→ Il suo dosaggio permette quindi di avere **informazioni** sull'**attività secretoria beta pancreatica** e di misurare così il tasso di **insulina endogena prodotta**

Dosaggio Immunochimico

valori di riferimento siero → 0.9-4.0 ng/ml

valori di riferimento urine → 30-180 ug/24h

Dosaggio utile nei **pazienti in terapia insulinica**, per **verificare la capacità residua** delle cellule beta a **produrre insulina endogena**.

C-Peptide

Livelli aumentati:
- Diabete 2 (non insulino-dipendente)
- Insulinoma
- **Insulino-resistenza**
- Insufficienza renale e Cushing
- Ipopotassiemia
- Ipoglicemizzanti orali
- Gravidanza
- In corso di **OGTT**

Livelli diminuiti:
- Diabete tipo 1
- Ipoglicemia factitia
- Diabete 2 in fase avanzata
- Pancreatectomia
- Diuretici e Alcool

INDICE HOMA-ß
(Homeostasis Model Assessment - ß cell function)

$$\text{Homa-ß} = \frac{20 \times \text{Insulinemia (mcU/ml)}}{\text{Glicemia (mmoli/l)} - 3.5} \%$$

Modello matematico di Mattheus
Fornisce una stima numerica e qualitativa sulla funzionalità delle cellule ß-pancreatiche

DIABETICMedicine

DOI: 10.1111/dme.12159

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Review Article

The clinical utility of C-peptide measurement in the care of patients with diabetes

A. G. Jones[1,2] and A. T. Hattersley[1,2]

[1]NIHR Exeter Clinical Research Facility, University of Exeter Medical School and [2]Diabetes and Endocrinology, Royal Devon and Exeter NHS Foundation Trust, Exeter, UK

Accepted 14 February 2013

Utilità clinica
Dosaggio del C-peptide nel diabete

Abstract

C-peptide is produced in equal amounts to insulin and is the best measure of endogenous insulin secretion in patients with diabetes. Measurement of insulin secretion using C-peptide can be helpful in clinical practice: differences in insulin secretion are fundamental to the different treatment requirements of Type 1 and Type 2 diabetes. This article reviews the use of C-peptide measurement in the clinical management of patients with diabetes, including the interpretation and choice of C-peptide test and its use to assist diabetes classification and choice of treatment. We provide recommendations for where C-peptide should be used, choice of test and interpretation of results. With the rising incidence of Type 2 diabetes in younger patients, the discovery of monogenic diabetes and development of new therapies aimed at preserving insulin secretion, the direct measurement of insulin secretion may be increasingly important. Advances in assays have made C-peptide measurement both more reliable and inexpensive. In addition, recent work has demonstrated that C-peptide is more stable in blood than previously suggested or can be reliably measured on a spot urine sample (urine C-peptide:creatinine ratio), facilitating measurement in routine clinical practice. The key current clinical role of C-peptide is to assist classification and management of insulin-treated patients. Utility is greatest after 3–5 years from diagnosis when persistence of substantial insulin secretion suggests Type 2 or monogenic diabetes. Absent C-peptide at any time confirms absolute insulin requirement and the appropriateness of Type 1 diabetes management strategies regardless of apparent aetiology.

Diabetes Ther (2017) 8:475–487
DOI 10.1007/s13300-017-0265-4

REVIEW

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CrossMark

A Practical Review of C-Peptide Testing in Diabetes

Emma Leighton · Christopher AR Sainsbury · Gregory C. Jones

Dosaggio del C-peptide nel diabete

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5446389/

Received: March 23, 2017 / Published online: May 8, 2017

ABSTRACT

C-peptide is a widely used measure of pancreatic beta cell function. It is produced in equimolar amounts to endogenous insulin but is excreted at a more constant rate over a longer time. Methods of estimation include urinary and unstimulated and stimulated serum sampling. Modern assays detect levels of c-peptide which can be used to guide diabetes diagnosis and

Keywords: C-peptide; Diabetes; Insulin; Type 1 diabetes; Type 2 diabetes

INTRODUCTION

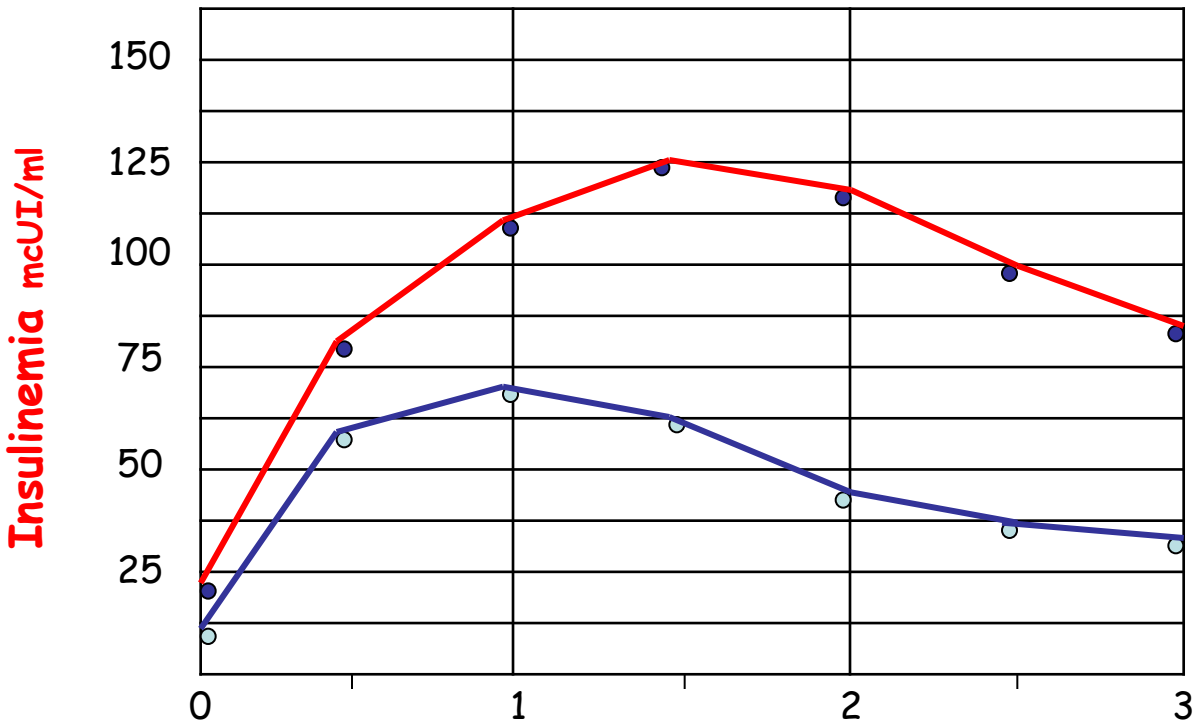
C-peptide is the part of proinsulin which is cleaved prior to co-secretion with insulin from pancreatic beta cells. Produced in equimolar amounts to endogenous insulin, it is not a product of therapeutically administered exoge-

Mauro Mario Amato

Curva Insulinemica da Carico orale di Glucosio

Si associa spesso alla OGTT per ottenere **informazioni sulla insulino-resistenza**.

- Determinazione Insulinemia a digiuno (**insulina base**).
- Somministrazione orale di **75 gr** di glucosio in soluzione con 200/250 ml di acqua (in caso soggetti con peso inferiore a 45 kg la quantità sarà di 1.75 gr./kg corporeo); tale soluzione bevuta con tranquillità entro 2/4 minuti.
- Prelievi ematici eseguiti a **30', 60', 90', 120' con determinazione Insulinemica**.
- I tempi possono variare, a seconda delle necessità individuali rilevate dal clinico, da un solo prelievo dopo 2 ore, a prelievi seriati che possono arrivare anche a 180'.



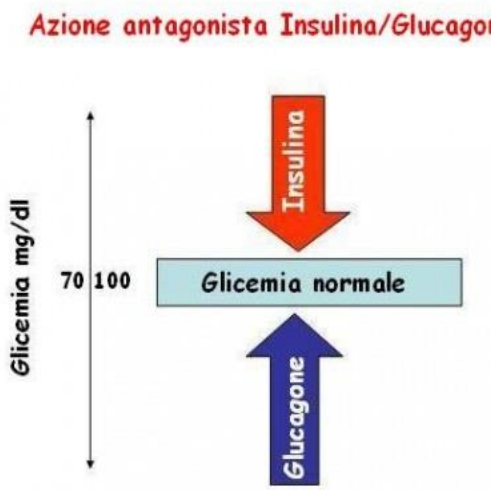
Normale

Insulino-resistenza (pre-diabete): Risposta aumentata con picco ritardato (90'-120')

Le curve riportate sono **andamenti tipici**. Non ci sono ben precisi valori di riferimento in quanto la **risposta insulinemica al carico può variare in relazione a diversi fattori individuali** (come età, sesso, condizioni fisio-patologiche) a conoscenza del **clinico che valuterà poi l'andamento generale della curva stessa**.

E' il **valore basale di insulinemia** che costituisce **elemento di riferimento**

METABOLISMO GLUCIDICO: Il Glucagone



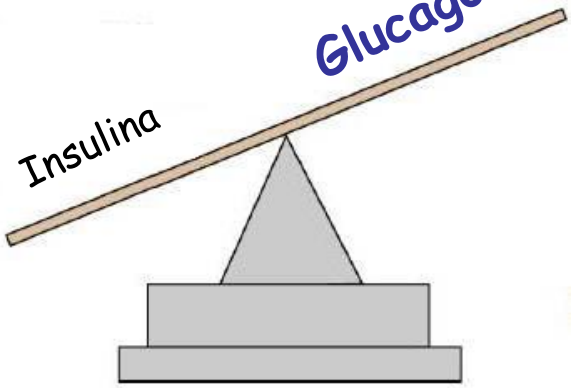
- **Ormone proteico** secreto dalle cellule alfa del pancreas endocrino ad azione antagonista verso l'insulina (**iperglicemizzante**): il valore della glicemia deriva dall'azione combinata di tali ormoni.
- **Diminuzione della glicemia** stimola alla sua secrezione per **attivare la glicogenolisi** e la **neoglucogenesi epatica**.
- **Non interviene sulla glicogenolisi muscolare** a differenza dell'adrenalina.
- Favorisce l'**ossidazione degli acidi grassi** dal tessuto adiposo inibendone la produzione.
- **Stimolano la sua produzione**:
  - Digiuno ed esercizio fisico intenso o prolungato
  - Concentrazione significativa di aminoacidi (in particolare arginina)
  - Cortisolo, catecolamine e GH
  - Ormoni gastroenterici (gastrina)
- **Inibiscono la sua secrezione**:
  - Aumento di acidi grassi circolanti
  - Somatostatina e secretina
  - Iperglicemia

Rapporto Insulina/Glucagone

Riveste **importantanza il loro rapporto**.
- Di norma è intorno a **2**
- Dopo un pasto può arrivare fino a **50-60**
- Dopo digiuno prolungato/esercizio intenso **0.5**
- Con rapporto **elevato** prevale lo stato **anabolico**
- Con rapporto **basso** prevale lo stato **catabolico**.

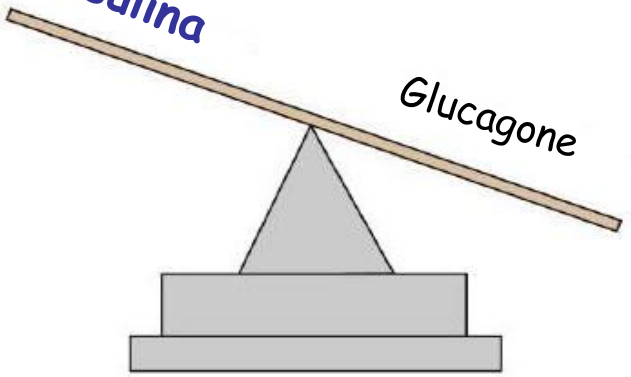
Con **pasto misto** aumenta l'**insulina**
Con **pasto proteico** aumenta il **glucagone**

METABOLISMO GLUCIDICO: Il Glucagone



- Glicogenolisi
- Neoglucogenesi
- Chetogenesi

- Glicolisi
- Sintesi Glicogeno
- Sintesi Lipidica
- Sintesi Proteica



Glucagone

Livelli aumentati:
- Digiuno ed esercizio fisico prolungato
- Diabete mellito scompensato
- Pancreatiti
- Insufficienza renale cronica
- Infezioni e Morbo di Cushing

Livelli diminuiti:
- Fibrosi cistica
- Pancreatica cronica
- Neoplasie pancreas
- Rimozione chirurgica pancreas

Dosaggio Immunochimico

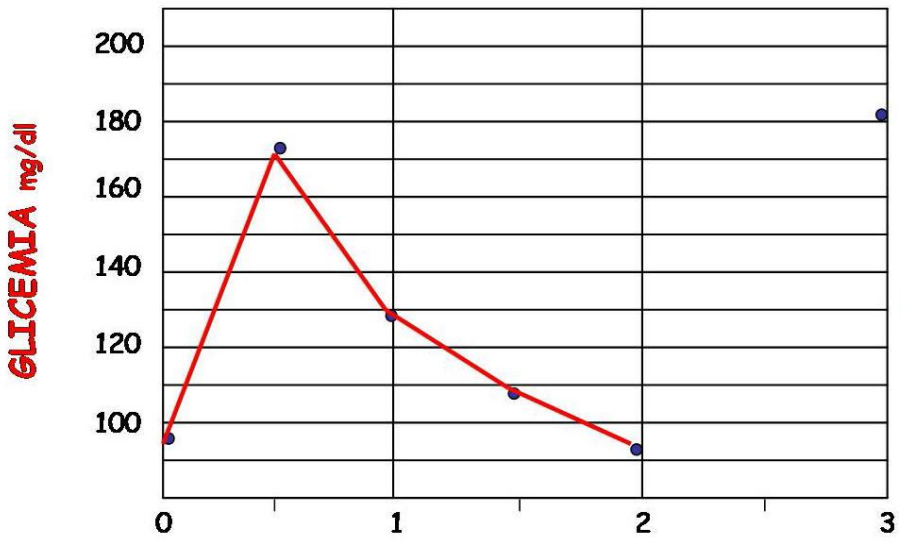
valori di riferimento → **40-180 pg/ml**

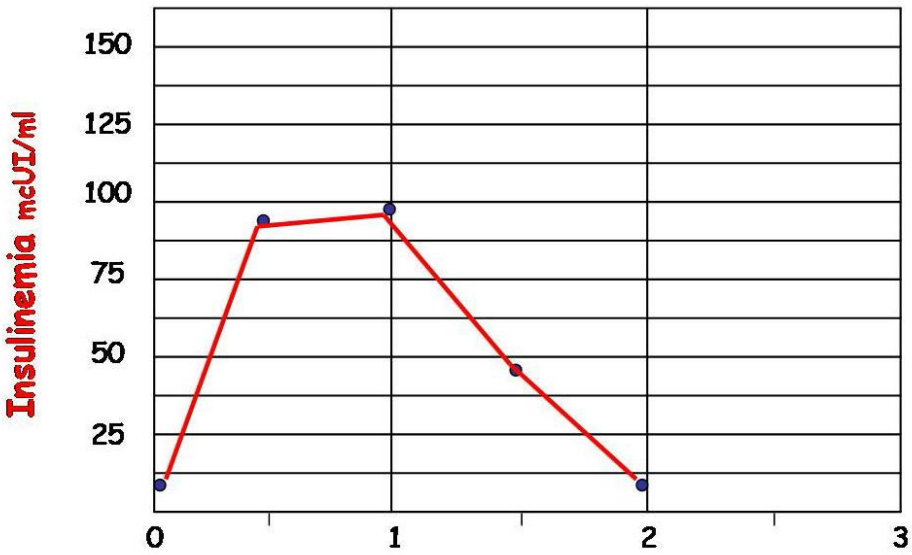
Chimica clinica

CURVA GLICEMICA DA CARICO © Enzimatico	mg/dl				
A DIGIUNO	**86** mg/dl	da	60	a	110
A 30 MINUTI DAL CARICO	**173** mg/dl	< 180			
A 60 MINUTI DAL CARICO	**128** mg/dl	< 180			
A 90 MINUTI DAL CARICO	**108** mg/dl	< 180			
A 120 MINUTI DAL CARICO	**74** mg/dl	< 153			

Dosaggi ormonali

INSULINA BASALE A DIGIUNO	**10.9** mcU/ml	da	3	a	25
INSULINA A 30 MINUTI	**94.3** mcU/ml	da	35	a	200
INSULINA A 60 MINUTI	**95.3** mcU/ml	da	40	a	200
INSULINA A 90 MINUTI	**45.9** mcU/ml	da	27	a	180
INSULINA A 120 MINUTI	**10.4** mcU/ml	da	15	a	150





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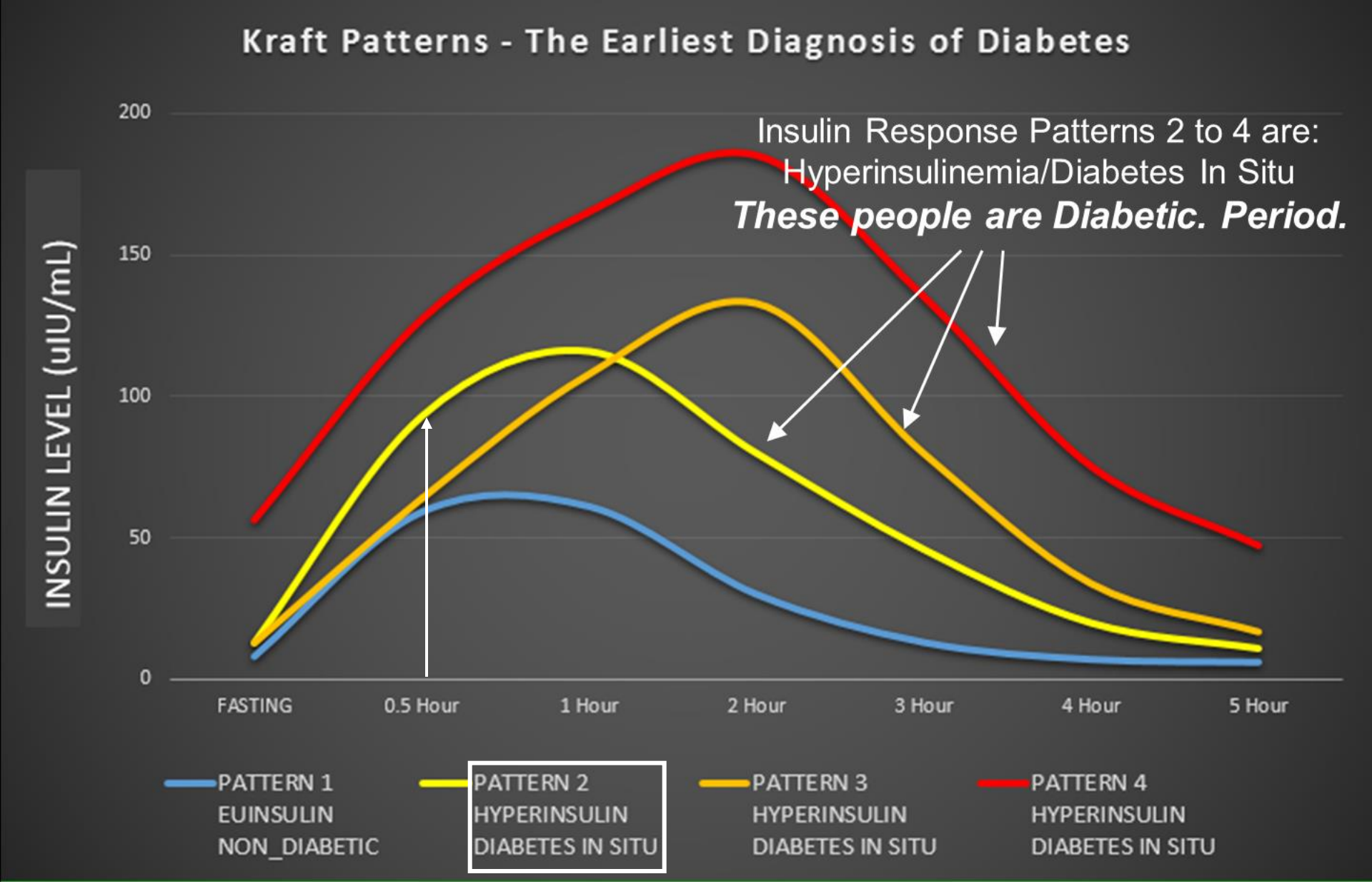

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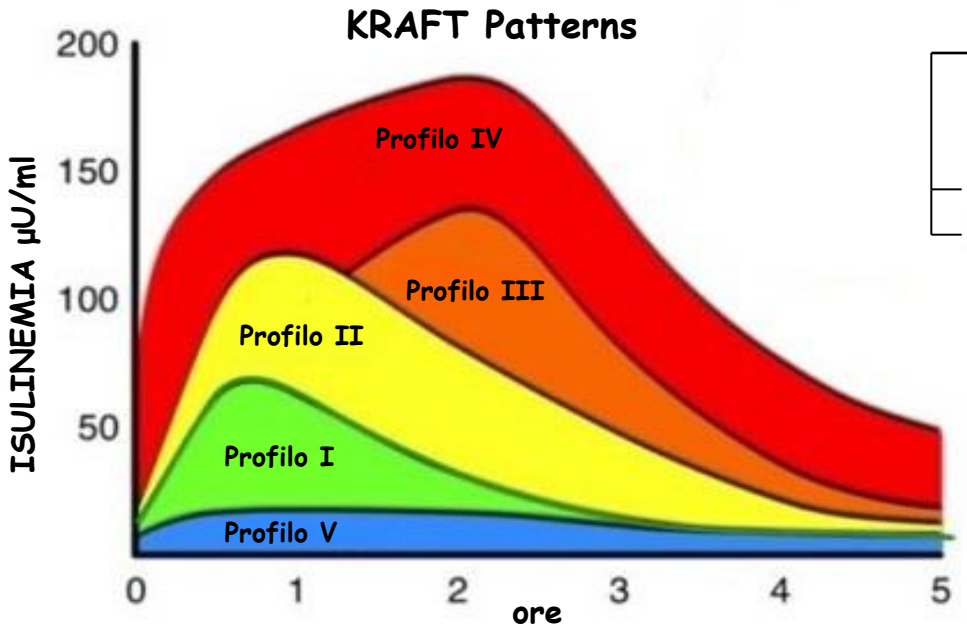
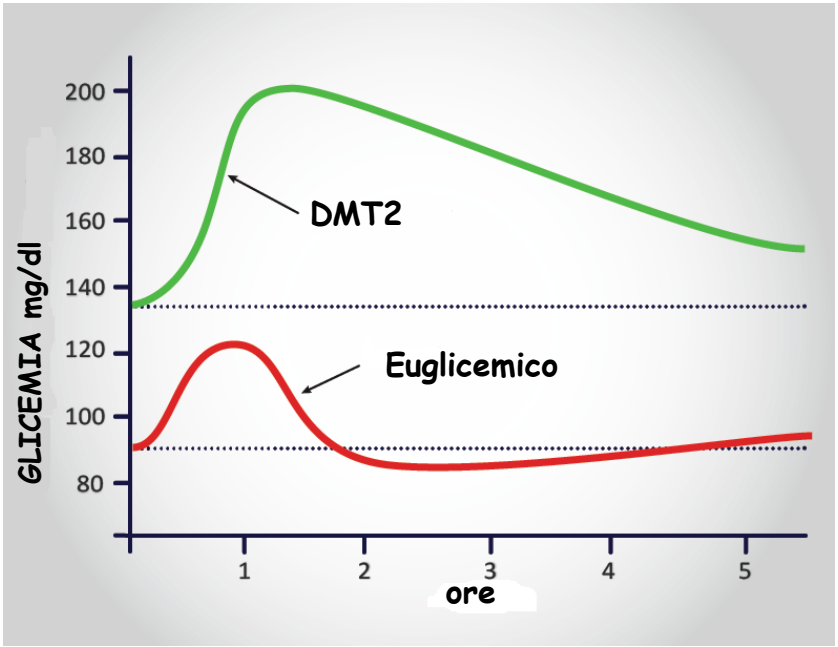


- Glicemia a digiuno nornale

Profilo I	Euglicemico	
Profilo II	Iperinsulinemia (rischio diabete 2 ↑)	
Profilo III	Iperinsulinemia (rischio diabete 2 ↑↑)	
Profilo IV	Iperinsulinemia (diabete in situ)	
Profilo V	Ipoinsulinemia	(diabete 1)



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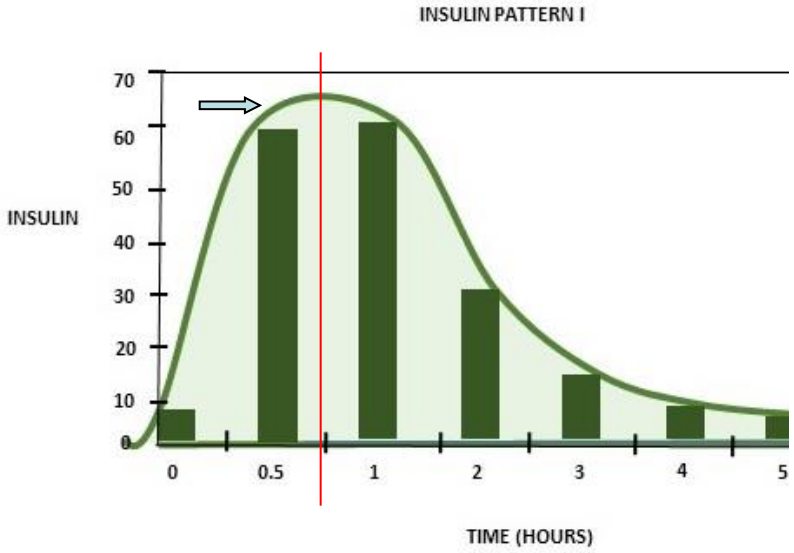
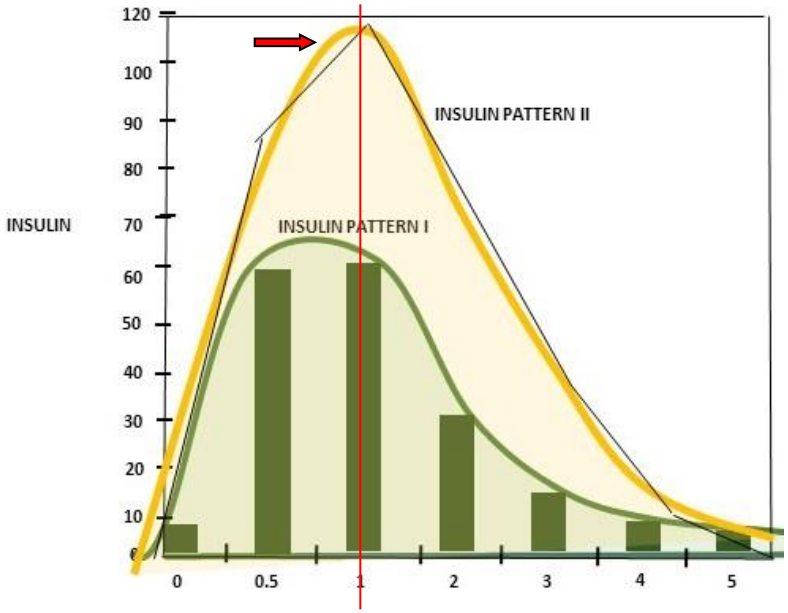
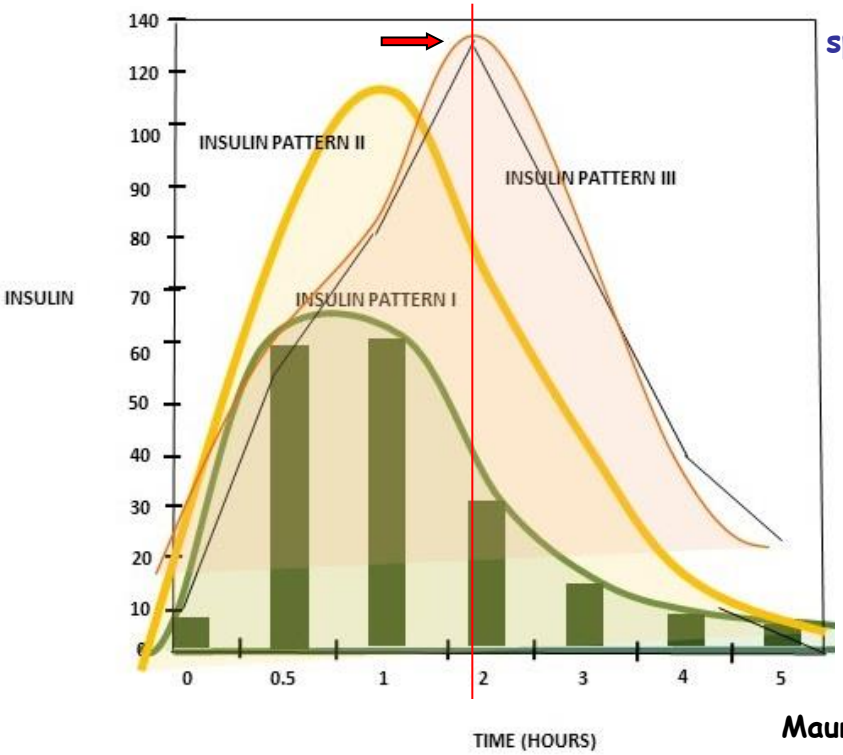
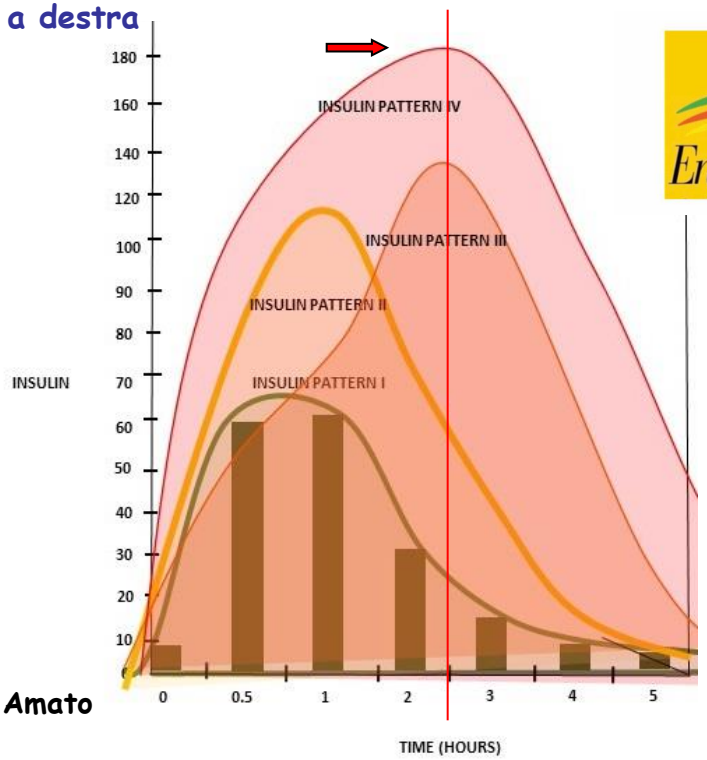




Picco più alto spostato a destra





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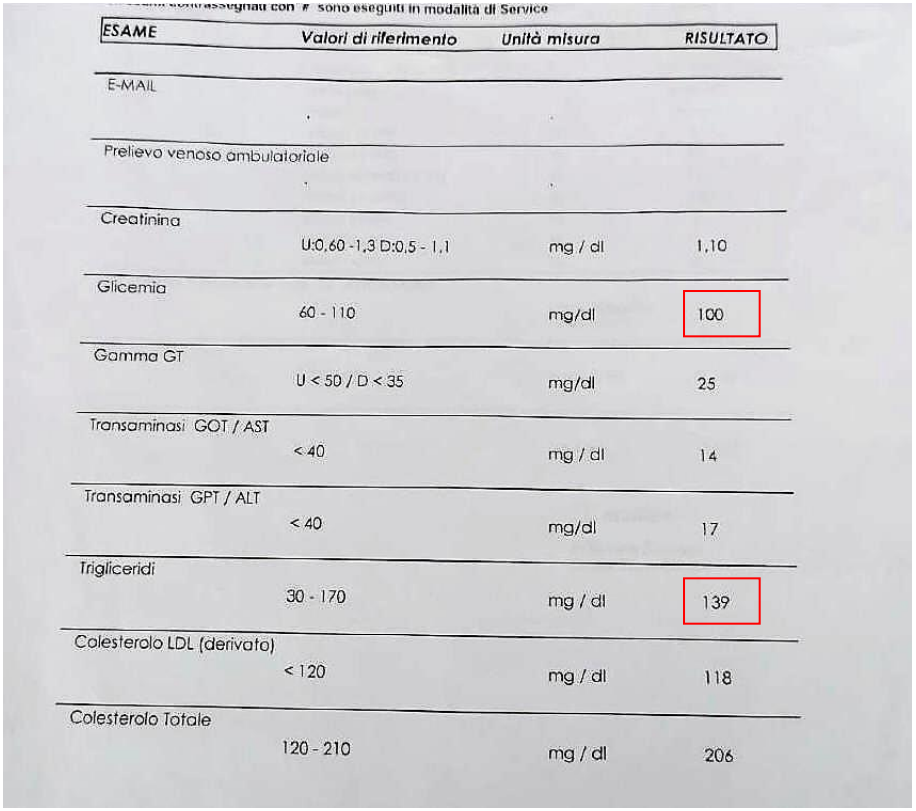
LABORATORIO E MATEMATICA
Resistenza Insulinica

- **Clamp Euglicemico-iperinsulinemico**
  (Test gold-standard abbastanza invasivo)
- **Curva Insulinemica da carico orale di glucosio**

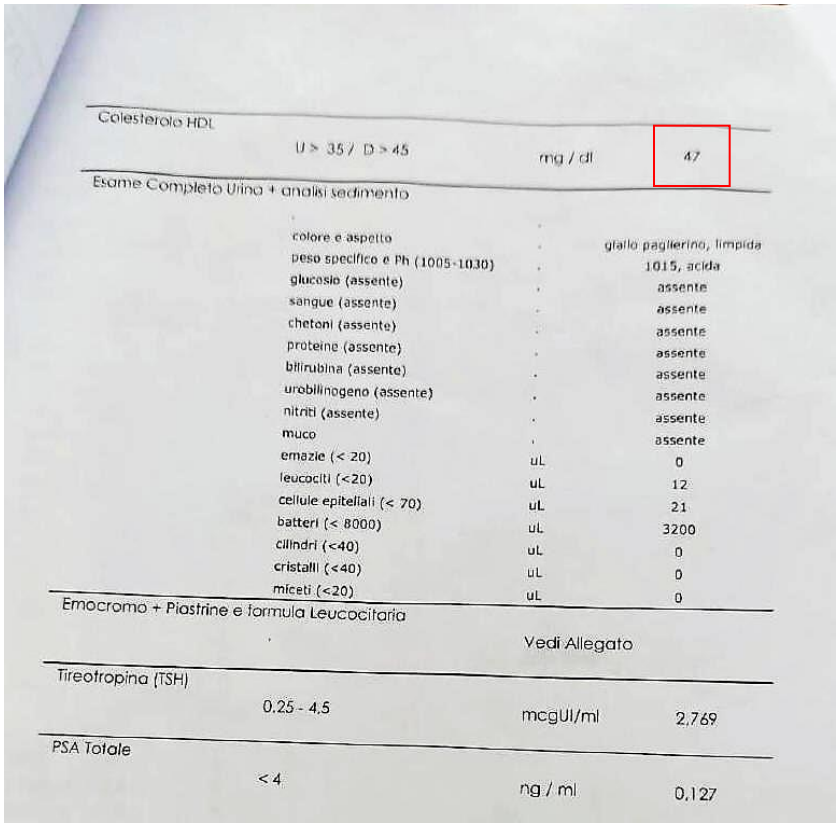
HOMA-IR (Homeostasis Model Assessment – Insulin Resistance)
HOMA-β (Homeostasis Model Assessment – β cell function)
Indice QUICKI (Quantitative Insulin sensitivity Check Index)

→ **Funzioni basate su Glicemia e Insulinemia a digiuno**

InsuTAG
TyG Index (Indice Trigliceridi-Glucosio)
Trigliceridi/HDL Ratio (Rapporto Trigliceridi-HDL-Colesterolo a digiuno)



ESAME	Valori di riferimento	Unità misura	RISULTATO
E-MAIL			
Prelievo venoso ambulatoriale			
Creatinina	U:0,60 -1,3 D:0,5 - 1,1	mg / dl	1,10
Glicemia	60 - 110	mg/dl	100
Gamma GT	U < 50 / D < 35	mg/dl	25
Transaminasi GOT / AST	< 40	mg / dl	14
Transaminasi GPT / ALT	< 40	mg/dl	17
Trigliceridi	30 - 170	mg / dl	139
Colesterolo LDL (derivato)	< 120	mg / dl	118
Colesterolo Totale	120 - 210	mg / dl	206



Colesterolo HDL	U > 35 / D > 45	mg / dl	47
Esame Completo Urina + analisi sedimento			
colore e aspetto			giallo paglierino, limpida
peso specifico e Ph (1005-1030)			1015, acida
glucosio (assente)			assente
sangue (assente)			assente
chetoni (assente)			assente
proteine (assente)			assente
bilirubina (assente)			assente
urobilinogeno (assente)			assente
nitriti (assente)			assente
muco			assente
emazie (< 20)		uL	0
leucociti (<20)		uL	12
cellule epiteliali (< 70)		uL	21
batteri (< 8000)		uL	3200
cilindri (<40)		uL	0
cristalli (<40)		uL	0
miceti (<20)		uL	0
Emocromo + Piastrine e formula Leucocitaria		Vedi Allegato	
Tireotropina (TSH)	0,25 - 4,5	mcgUI/ml	2,769
PSA Totale	< 4	ng / ml	0,127

TyG Index (Indice Trigliceridi-Glucosio)

Trigliceridi/HDL Ratio (Rapporto Trigliceridi-HDL-Colesterolo a digiuno)

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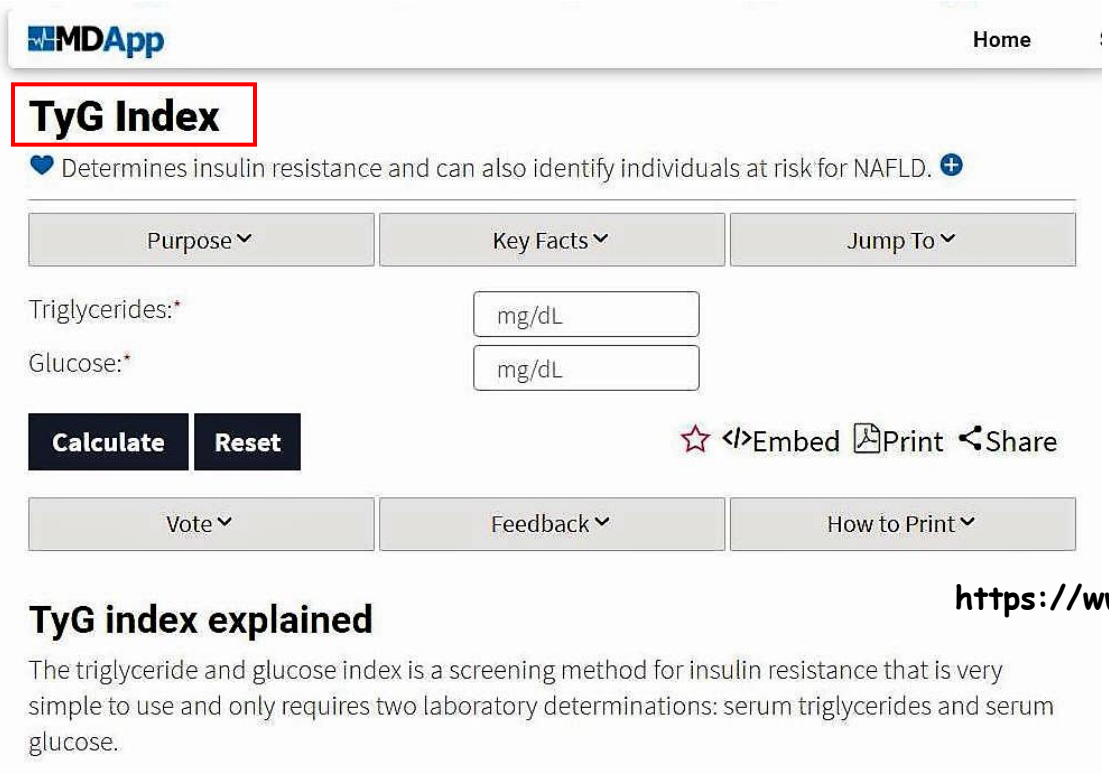
TyG Index
Indice Trigliceridi-Glucosio

- Sembra avere anch'esso buona correlazione con l'**insulino-resistenza nei soggetti con sindrome metabolica**
- Può essere usato anche come **indice di rigidezza delle arterie** e come predittore dell'**incidenza** della **NAFLD**

TyG Index = **Log** [**Trigliceridi** (mg/dl) x **Glicemia** (mg/dl) **/ 2**]

borderline **4.3**



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https://www.mdapp.co/tyg-index-calculator-359/

TRIGLYCERIDES/HDL Ratio

Indice Trigliceridi-HDL

Trigliceridi/HDL Ratio= **Trigliceridi** (mg/dl) **/ HDL-Colesterolo** (mg/dl)

cut-off **1.1 – 1.8**

- Consente **stima** dell'**insulino-resistenza**
- Può fornire indicazioni sulla **resistenza insulinica anche nei soggetti obesi, nel sovrappeso pediatrico e cancro correlata**
- Viene usato inoltre come **marker di rischio per diabete tipo 2** e malattie cardiovascolari

Glicemia	**100 mg/dl**
Colesterolo totale	**206 mg/dl**
HDL-Colesterolo	**47 mg/dl**
Trigliceridi	**139 mg/dl**

Indice Trigliceridi/HDL = **139** (mg/dl) **/ 47** (mg/dl) = **2.96**

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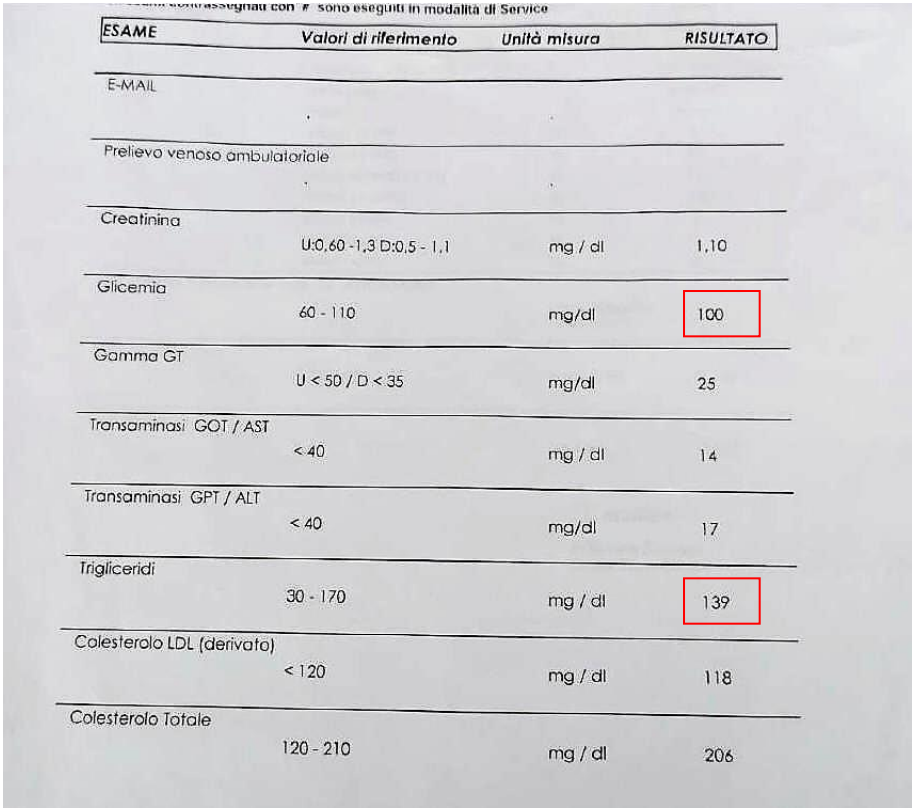

sono eseguiti in modalità di Service

ESAME	Valori di riferimento	Unità misura	RISULTATO
E-MAIL			
Prelievo venoso ambulatoriale			
Creatinina	U:0,60 -1,3 D:0,5 - 1,1	mg / dl	1,10
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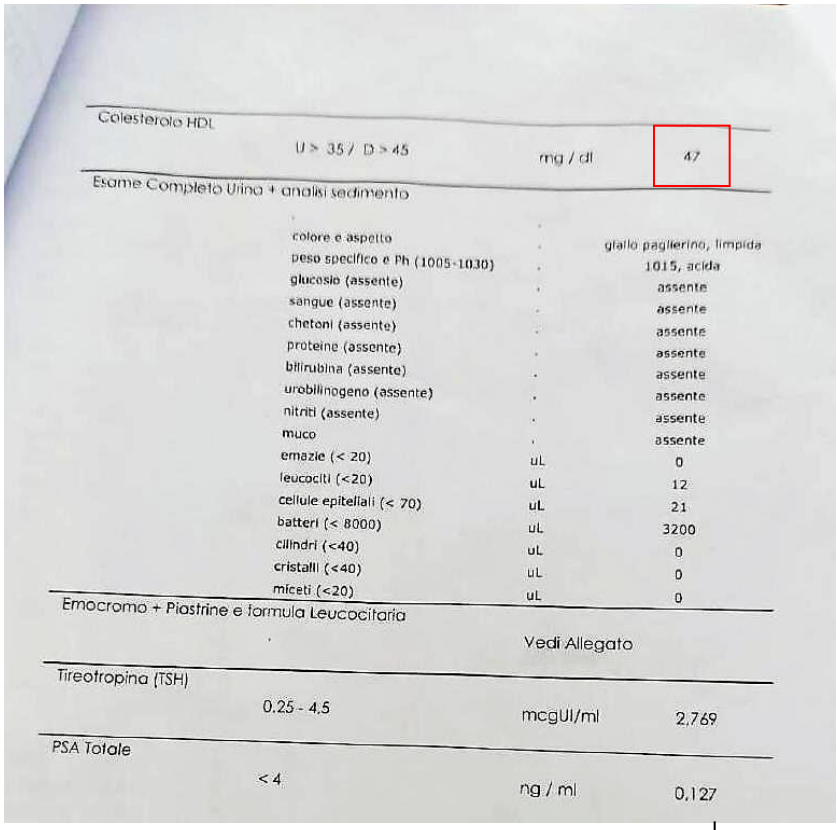

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peso specifico e Ph (1005-1030)			1015, acida
glucosio (assente)			assente
sangue (assente)			assente
chetoni (assente)			assente
proteine (assente)			assente
bilirubina (assente)			assente
urobilinogeno (assente)			assente
nitriti (assente)			assente
muco			assente
emazie (< 20)		uL	0
leucociti (<20)		uL	12
cellule epiteliali (< 70)		uL	21
batteri (< 8000)		uL	3200
cilindri (<40)		uL	0
cristalli (<40)		uL	0
miceti (<20)		uL	0
Emocromo + Piastrine e formula Leucocitaria		Vedi Allegato	
Tireotropina (TSH)	0,25 - 4,5	mcgUI/ml	2,769
PSA Totale	< 4	ng / ml	0,127

TyG Index (Indice Trigliceridi-Glucosio)

Trigliceridi/HDL Ratio (Rapporto Trigliceridi-HDL-Colesterolo a digiuno)

Insulino-resistenza latente con rischio di DMT2 ← interpretazione

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INDICE HOMA-IR
(Homeostasis Model Assessment – Insulin Resistance)

$$\text{Homa-IR} = \frac{\text{Glicemia (mg/dl)} \times \text{Insulinemia (mcU/ml)}}{405}$$

$$\text{Homa-IR} = \frac{\text{Glicemia (mmoli/l)} \times \text{Insulinemia (mU/l)}}{22.5}$$

Valori di riferimento { **0.22 – 2.4** (**mediana 1.21**)
fino a 8-10 anni **0.20-3.4**

- Deriva da un modello matematico di Mattheus e **fornisce** una **stima numerica e qualitativa** sulla resistenza insulinica basale.
- Tale indice si dimostra molto **utile per grandi studi epidemiologici**.
- **Buona correlazione** con i dati ottenuti con misurazioni dirette nelle seguenti **condizioni**:

→ Prelievo eseguito su **due provette separate** dopo digiuno di 8-9 ore.

-Glicemia e insulinemia devono essere **eseguiti su siero in un'unica seduta**.
- L'**HOMA** non può essere eseguito dopo intensa attività fisica o dopo aver fumato (anche una sigaretta).

- Tale indice deve essere valutato nel contesto di altri dati antropometrici e BMI.
- Si preferisce talvolta valutare l'insulino-resistenza con la **curva insulinemica da carico orale di glucosio in contemporanea OGTT**.

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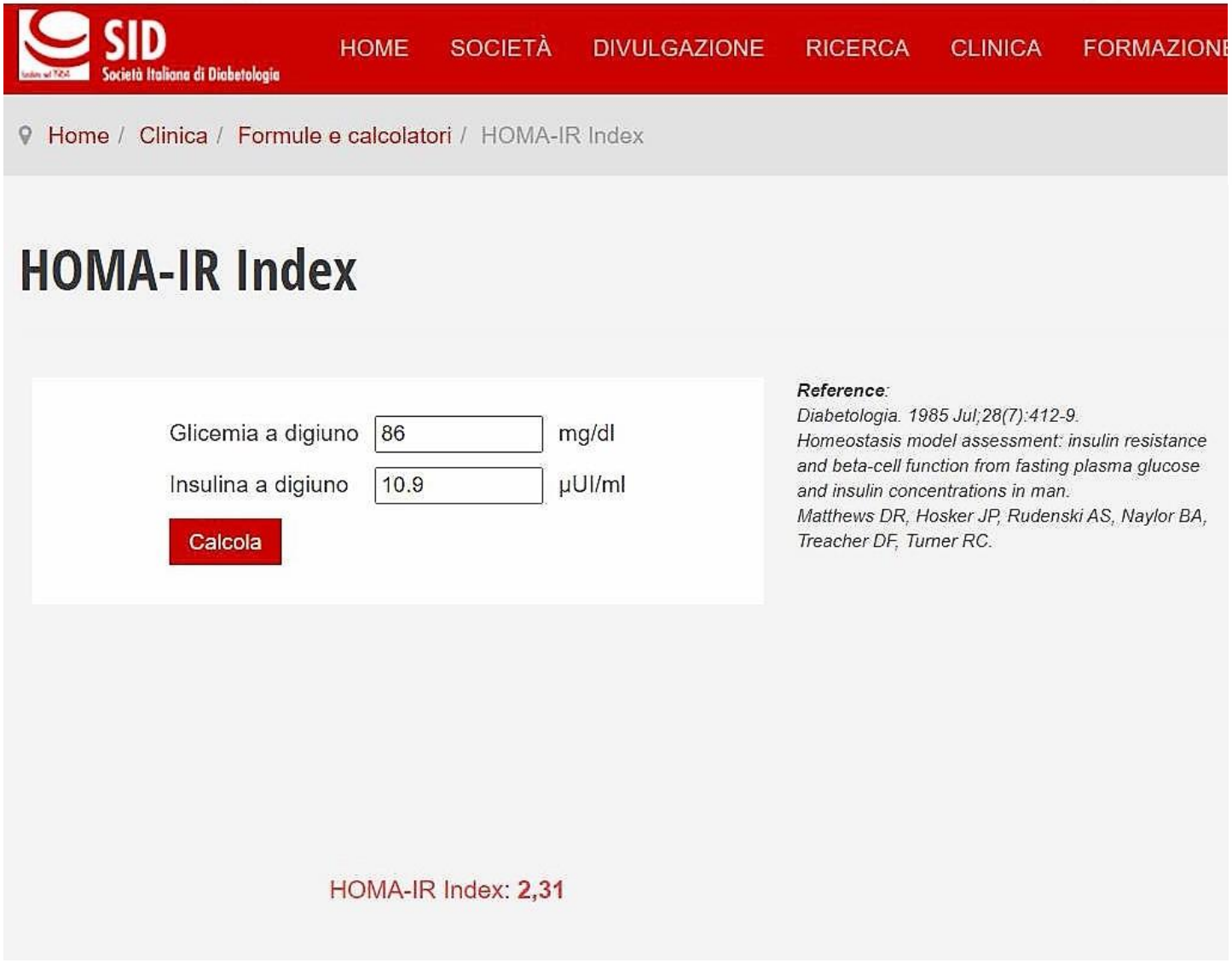


http://www.siditalia.it/clinica/formule-e-calcolatori/homa-ir-index

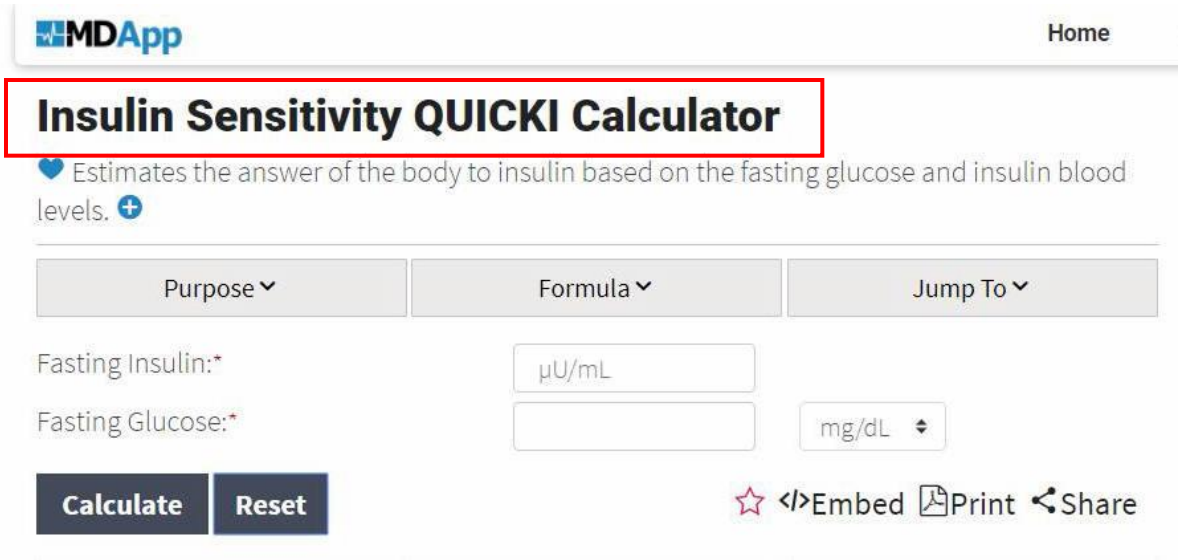
INDICE QUICKI
(Quantitative Insulin sensitivity Check Index)

$$\frac{1}{\text{Log(Insulina } \mu\text{U/ml)} + \text{Log(Glucosio mg/dl)}}$$

Modello matematico di Katz che **fornisce** una **stima numerica e qualitativa** sulla **sensibilità insulinica** basale (**dosaggio Insulina e Glicemia a digiuno**)

valori **inferiori a 0,34** indicano la **insulino-resistenza**

valori =< **0.30** **diabete**



https://www.mdapp.co/insulin-sensitivity-quicki-calculator-324/

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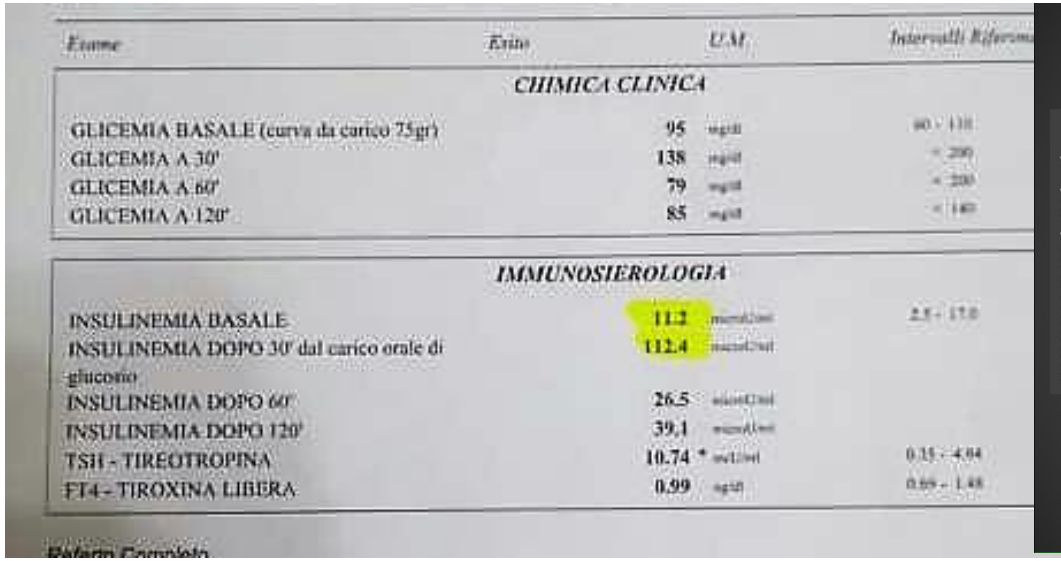
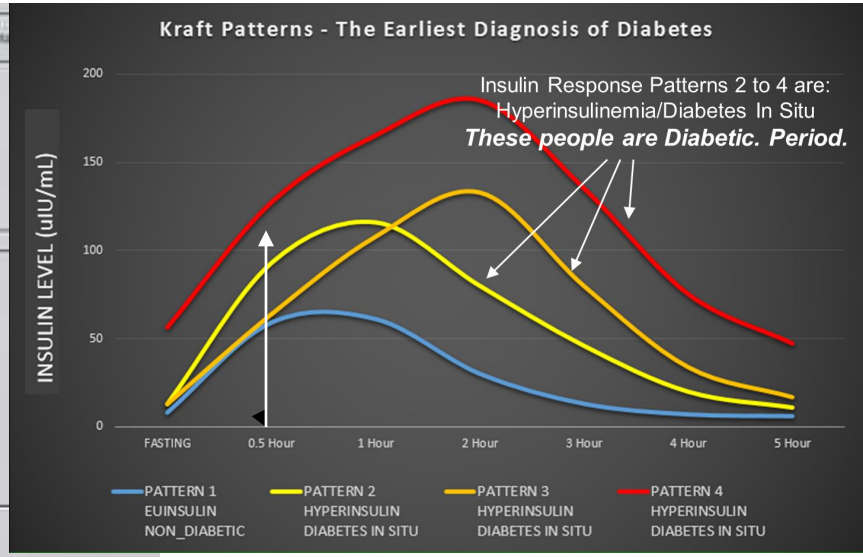


Esame	Esito	U.M.	Intervalli Riferimento
CHIMICA CLINICA			
GLICEMIA BASALE (curva da carico 75gr)	95	mg/dl	60 - 110
GLICEMIA A 30'	138	mg/dl	< 200
GLICEMIA A 60'	79	mg/dl	< 200
GLICEMIA A 120'	85	mg/dl	< 140
IMMUNOSIEROLOGIA			
INSULINEMIA BASALE	11.2	microU/ml	2.5 - 17.0
INSULINEMIA DOPO 30' dal carico orale di glucosio	112.4	microU/ml	
INSULINEMIA DOPO 60'	26.5	microU/ml	
INSULINEMIA DOPO 120'	39,1	microU/ml	
TSH - TIREOTROPINA	10.74 *	uU/ml	0.35 - 4.94
FT4 - TIROXINA LIBERA	0.99	ng/dl	0.89 - 1.48

Referto Completo



Homa-IR= 2.63

0.22 – 2.4 (mediana 1.21)

INDICE QUICKI= 0.33

< 0,34 indicano
insulino-resistenza

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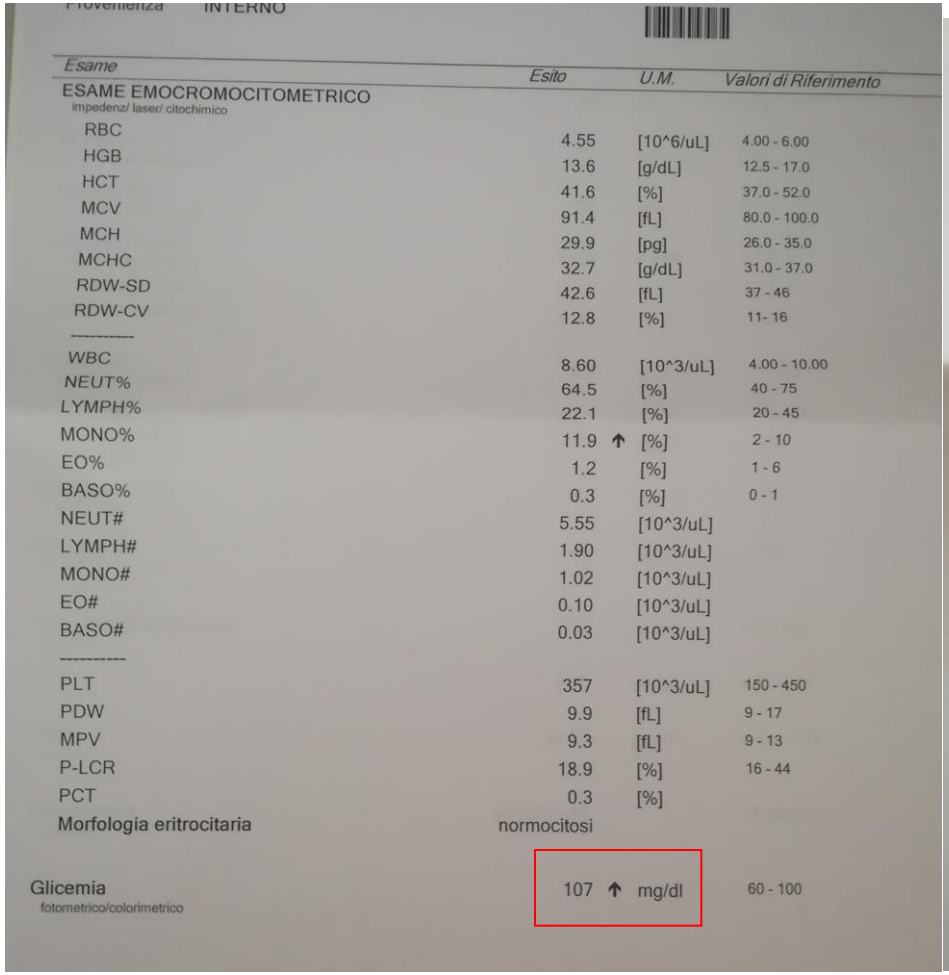
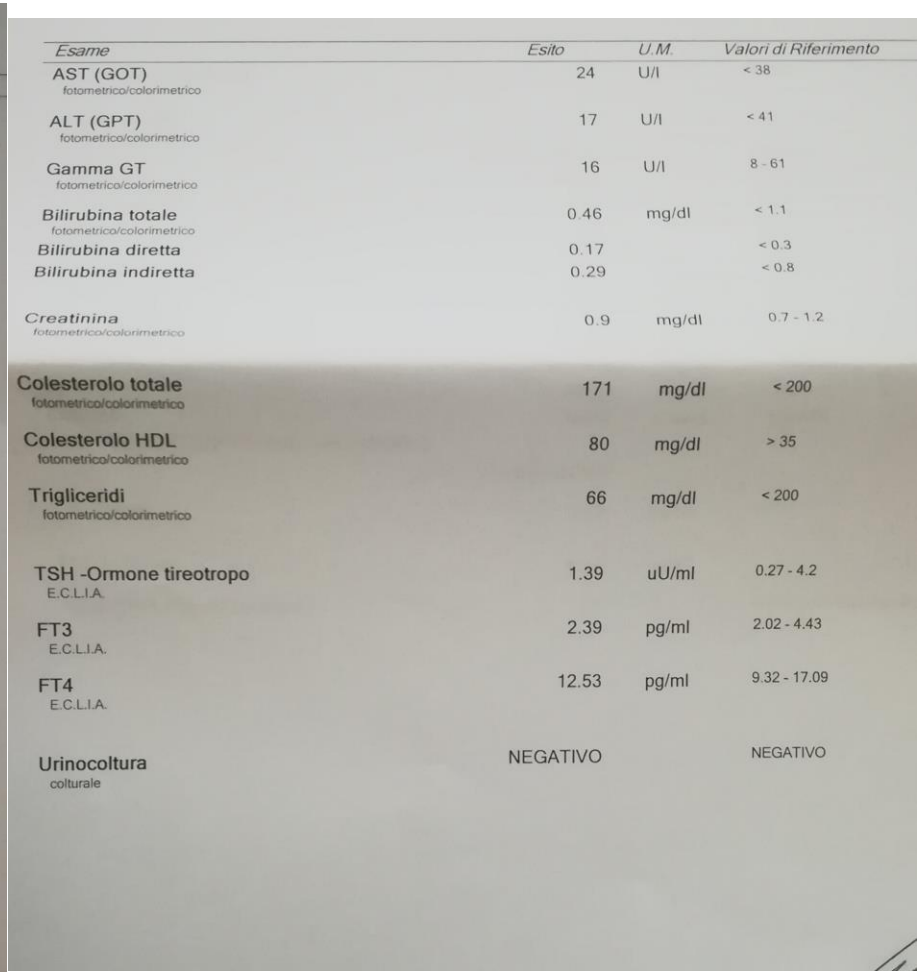


Provenienza INTERNO

Esame	Esito	U.M.	Valori di Riferimento
ESAME EMOCROMOCITOMETRICO (impedenz/ laser/ citochimico)			
RBC	4.55	[10^6/uL]	4.00 - 6.00
HGB	13.6	[g/dL]	12.5 - 17.0
HCT	41.6	[%]	37.0 - 52.0
MCV	91.4	[fL]	80.0 - 100.0
MCH	29.9	[pg]	26.0 - 35.0
MCHC	32.7	[g/dL]	31.0 - 37.0
RDW-SD	42.6	[fL]	37 - 46
RDW-CV	12.8	[%]	11- 16
WBC	8.60	[10^3/uL]	4.00 - 10.00
NEUT%	64.5	[%]	40 - 75
LYMPH%	22.1	[%]	20 - 45
MONO%	11.9 ↑	[%]	2 - 10
EO%	1.2	[%]	1 - 6
BASO%	0.3	[%]	0 - 1
NEUT#	5.55	[10^3/uL]	
LYMPH#	1.90	[10^3/uL]	
MONO#	1.02	[10^3/uL]	
EO#	0.10	[10^3/uL]	
BASO#	0.03	[10^3/uL]	
PLT	357	[10^3/uL]	150 - 450
PDW	9.9	[fL]	9 - 17
MPV	9.3	[fL]	9 - 13
P-LCR	18.9	[%]	16 - 44
PCT	0.3	[%]	
Morfologia eritrocitaria	normocitosi		
Glicemia (fotometrico/colorimetrico)	107 ↑	mg/dl	60 - 100



Esame	Esito	U.M.	Valori di Riferimento
AST (GOT) (fotometrico/colorimetrico)	24	U/l	< 38
ALT (GPT) (fotometrico/colorimetrico)	17	U/l	< 41
Gamma GT (fotometrico/colorimetrico)	16	U/l	8 - 61
Bilirubina totale (fotometrico/colorimetrico)	0.46	mg/dl	< 1.1
Bilirubina diretta	0.17		< 0.3
Bilirubina indiretta	0.29		< 0.8
Creatinina (fotometrico/colorimetrico)	0.9	mg/dl	0.7 - 1.2
Colesterolo totale (fotometrico/colorimetrico)	171	mg/dl	< 200
Colesterolo HDL (fotometrico/colorimetrico)	80	mg/dl	> 35
Trigliceridi (fotometrico/colorimetrico)	66	mg/dl	< 200
TSH -Ormone tireotropo (E.C.L.I.A.)	1.39	uU/ml	0.27 - 4.2
FT3 (E.C.L.I.A.)	2.39	pg/ml	2.02 - 4.43
FT4 (E.C.L.I.A.)	12.53	pg/ml	9.32 - 17.09
Urinocoltura (colturale)	NEGATIVO		NEGATIVO

TyG Index= 4.43 borderline **4.4**

Trigliceridi/HDL Ratio= 0.83 cut-off **1.1 – 1.8**

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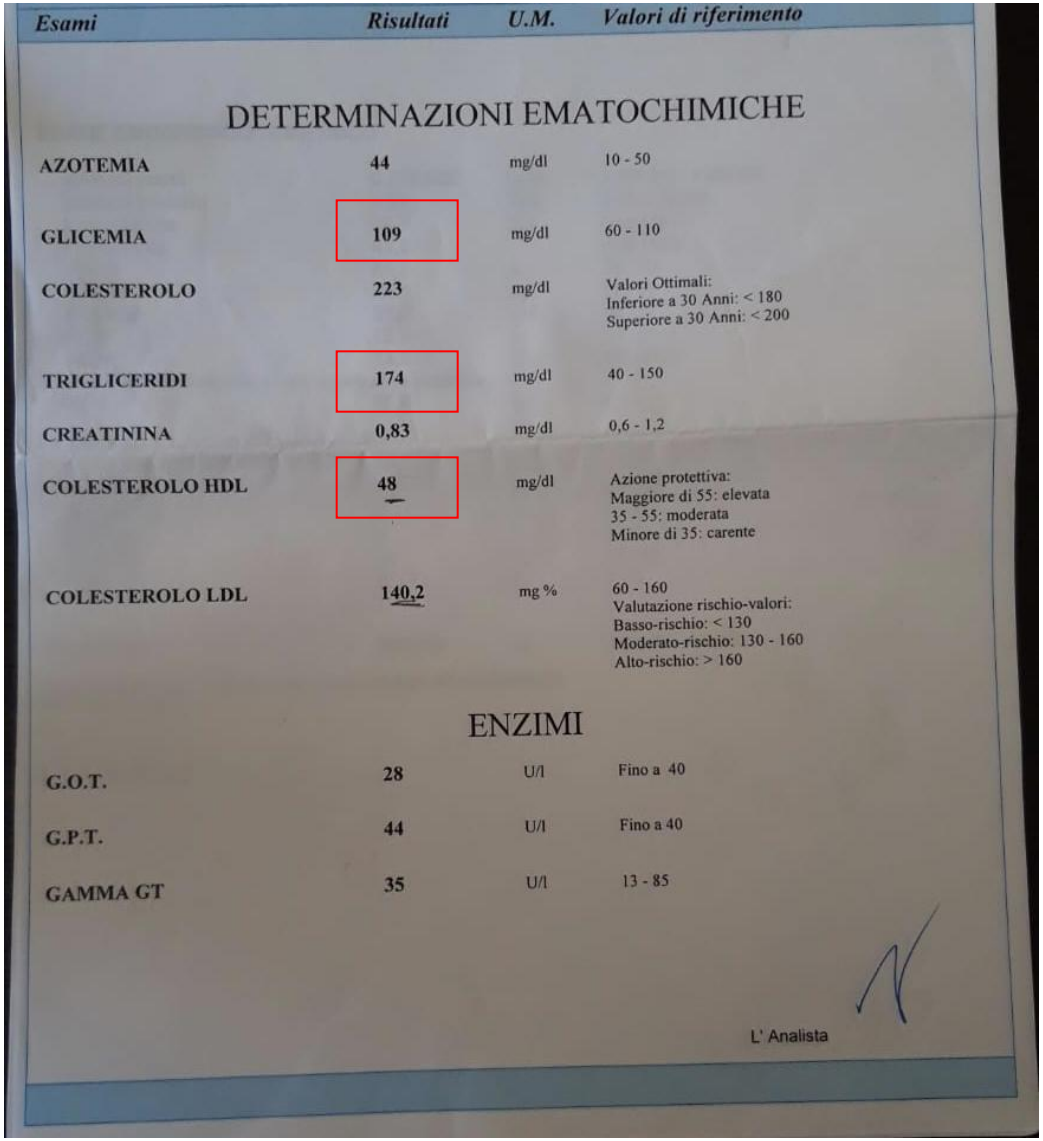
Mauro Mario Amato



Esami	Risultati	U.M.	Valori di riferimento
DETERMINAZIONI EMATOCHIMICHE			
AZOTEMIA	44	mg/dl	10 - 50
GLICEMIA	109	mg/dl	60 - 110
COLESTEROLO	223	mg/dl	Valori Ottimali: Inferiore a 30 Anni: < 180 Superiore a 30 Anni: < 200
TRIGLICERIDI	174	mg/dl	40 - 150
CREATININA	0,83	mg/dl	0,6 - 1,2
COLESTEROLO HDL	48	mg/dl	Azione protettiva: Maggiore di 55: elevata 35 - 55: moderata Minore di 35: carente
COLESTEROLO LDL	140,2	mg %	60 - 160 Valutazione rischio-valori: Basso-rischio: < 130 Moderato-rischio: 130 - 160 Alto-rischio: > 160
ENZIMI			
G.O.T.	28	U/l	Fino a 40
G.P.T.	44	U/l	Fino a 40
GAMMA GT	35	U/l	13 - 85

L' Analista

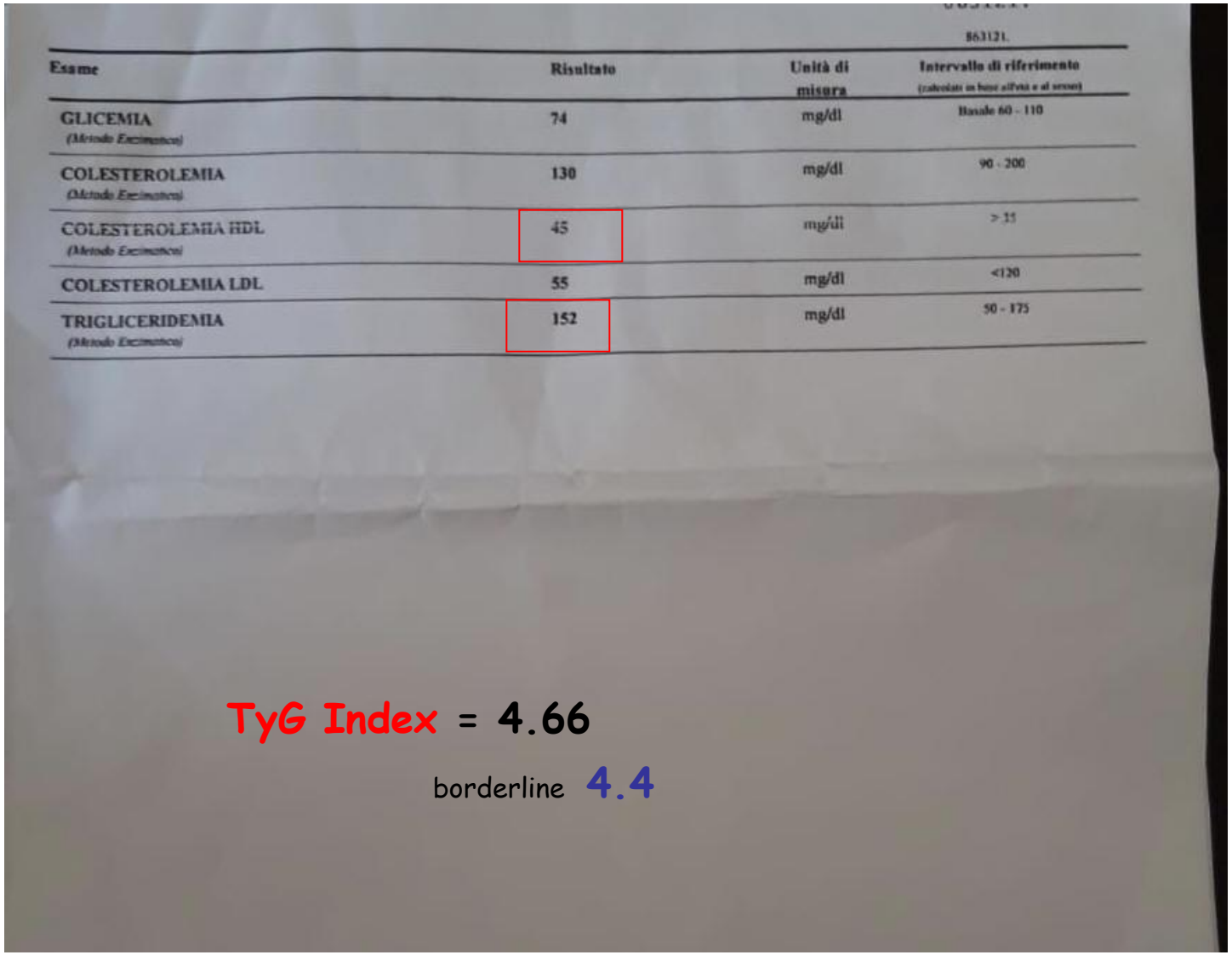
Enpab webinar

Insulino-resistenza
Rischio DMT2

Mauro Mario Amato

Indice Trigliceridi/HDL = **174** (mg/dl) **/ 48** (mg/dl) = **3.63** cut-off **1.1 – 1.8**

TyG Index= 4.93 borderline **4.4**



Esame	Risultato	Unità di misura	Intervallo di riferimento
GLICEMIA	74	mg/dl	Basale 60 - 110
COLESTEROLEMIA	130	mg/dl	90 - 200
COLESTEROLEMIA HDL	45	mg/dl	> 35
COLESTEROLEMIA LDL	55	mg/dl	<120
TRIGLICERIDEMIA	152	mg/dl	50 - 175

TyG Index = 4.66

borderline **4.4**

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Insulino-resistenza?

Mauro Mario Amato

Indice Trigliceridi/HDL = 152 (mg/dl) / 45 (mg/dl) = 3.38

cut-off **1.1 – 1.8**

Mauro Mario Amato

4° mese di gravidanza, 37 anni, ha iniziato la gravidanza in sovrappeso e al momento il peso è fermo perché nel primo trimestre ha avuto nausea gravidica. Ultimamente lamenta tachicardia. Da circa 1 mese assume Natalben. Visti i trigliceridi e la glicemia dovrebbe mettersi a dieta (è un'amica, non una paziente...quindi non mi ascolta) e a maggior ragione visto l'FT4. Non so se conferma... Mi piacerebbe avere un suo commento generale. Come interpreterebbe il valore della ferritina, anche in relazione alla microcitemia in seconda pagina? Va valutato semplicemente come indice infiammatorio?

DESCRIZIONE ESAME	RISULTATO	Unità misura	Valore normale
Chimica Clinica			
COLESTEROLO TOTALE *Metodo : Enzimatico colorimetrico*	247 *	mg/dl	150 - 200 Desiderabile
COLESTEROLO HDL *Metodo : Enzimatico colorimetrico*	71	mg/dl	> 60 Desiderabile
TRIGLICERIDI *Metodo : Enzimatico colorimetrico*	165	mg/dl	50 - 172
Anemia			
SIDEREMIA *Metodo : Colorimetrico*	78	µg/dl	49 - 151
FERRITINA *Metodo : Immunoturbidimetrico*	324,00 *	ng/ml	15.00 - 150.00
Tiroide			
FT4 LIBERO *Metodo : ECLIA*	0,81 *	ng/dl	0.93 - 1.7
Diabetologia			
GLICEMIA *Metodo: Fotometrico*	85	mg/dl	60 - 100

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Modesta Insulino-resistenza

↓

Sovrappeso fetale?

Mauro Mario Amato

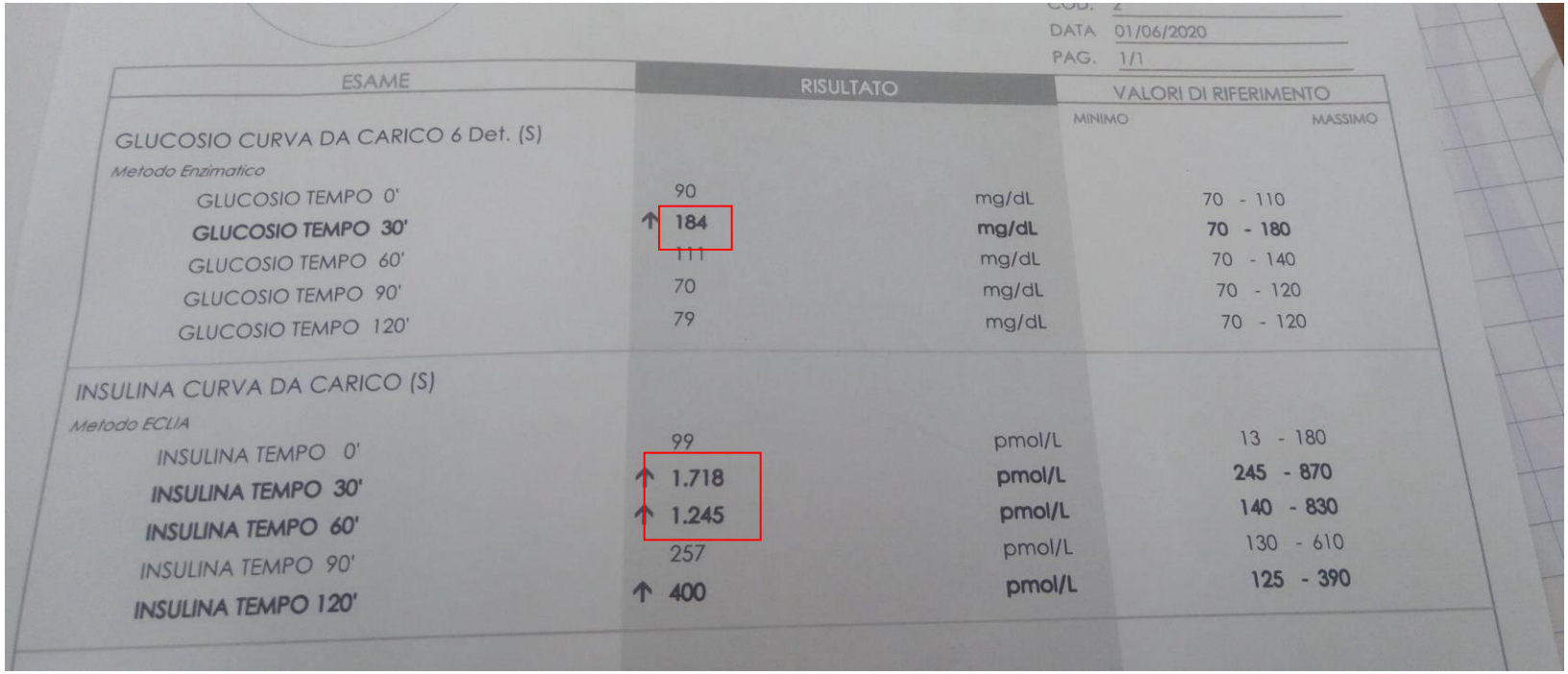
Indice Trigliceridi/HDL = 2.32

cut-off 1.1 – 1.8

TyG Index= 4.77

borderline 4.4

Mauro Mario Amato



DATA 01/06/2020
PAG. 1/1

ESAME	RISULTATO		VALORI DI RIFERIMENTO MINIMO	MASSIMO
GLUCOSIO CURVA DA CARICO 6 Det. (S)				
Metodo Enzimatico				
GLUCOSIO TEMPO 0'	90	mg/dL	70	110
GLUCOSIO TEMPO 30'	↑ **184**	**mg/dL**	**70**	**180**
GLUCOSIO TEMPO 60'	111	mg/dL	70	140
GLUCOSIO TEMPO 90'	70	mg/dL	70	120
GLUCOSIO TEMPO 120'	79	mg/dL	70	120
INSULINA CURVA DA CARICO (S)				
Metodo ECLIA				
INSULINA TEMPO 0'	99	pmol/L	13	180
INSULINA TEMPO 30'	↑ **1.718**	**pmol/L**	**245**	**870**
INSULINA TEMPO 60'	↑ **1.245**	**pmol/L**	**140**	**830**
INSULINA TEMPO 90'	257	pmol/L	130	610
INSULINA TEMPO 120'	↑ **400**	**pmol/L**	**125**	**390**

Insulina fc = 6.945

Base 14.2
30' 247
60' 179
90' 40
120' 58

Iperinsulinemico con diabete in situ

→ **Marcatori di metabolismo glucidico**

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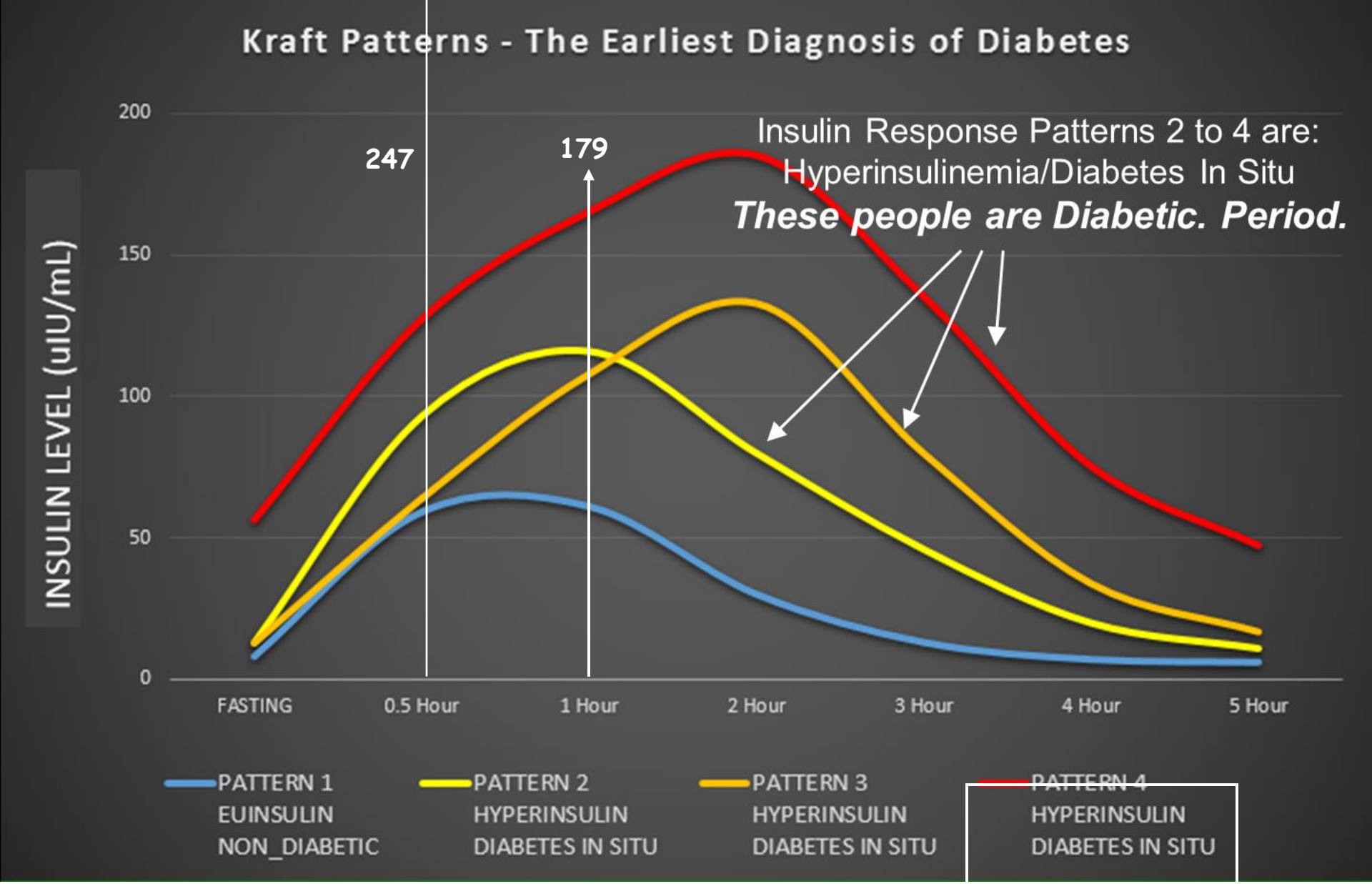
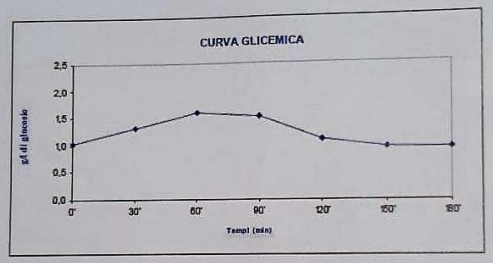

Kraft Patterns - The Earliest Diagnosis of Diabetes
INSULIN LEVEL (uIU/mL)
200
150
100
50
0
247
179
Insulin Response Patterns 2 to 4 are:
Hyperinsulinemia/Diabetes In Situ
These people are Diabetic. Period.
FASTING
0.5 Hour
1 Hour
2 Hour
3 Hour
4 Hour
5 Hour
PATTERN 1
EUINSULIN
NON_DIABETIC
PATTERN 2
HYPERINSULIN
DIABETES IN SITU
PATTERN 3
HYPERINSULIN
DIABETES IN SITU
PATTERN 4
HYPERINSULIN
DIABETES IN SITU

ESAME	Risultato	Unità di misura	Valori Riferimento
CURVA INSULINICA (dopo carico di glucosio)			
(Met. Chemiluminescenza)			
INSULINA 0'	19,1	mcU/ml	2,0 - 30,0
" + 30'	56,5	"	
" + 60'	70,3	"	
" + 90'	108,1	"	
" + 120'	114,5	"	
" + 150'	54,1	"	
" + 180'	19,0	"	

ESAME	Risultato	Unità di misura	Valori Riferimento
OMOCISTEINA (Met. Chemiluminescenza)	5,87	µmol/l	3,50 - 14,00
TSH (Ultra Sensibile) (Met. Chemiluminescenza)	1,210	mIU/l	0,270 - 4,200
CURVA GLICEMICA (dopo carico di glucosio)			
(Met. Enzimatico Colorimetrico)			
GLICEMIA 0'	1,03	g/l	0,6 - 1,10
" + 30'	1,32	"	
" + 60'	1,60	"	
" + 90'	1,52	"	
" + 120'	1,09	"	
" + 150'	0,95	"	
" + 180'	0,94	"	
GLICOSURIA + 120'	0,0	g/l	



Curva Insulina			
Insulina	17,00	µU/mL	3,0 - 17,0
Insulina (Prelievo 60 min.)	107,20	µU/mL	
Insulina (Prelievo 120 min.)	73,18	µU/mL	
Insulina (Prelievo 180 min.)	12,62	µU/mL	
Insulina (Prelievo 240 min.)	11,84	µU/mL	

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Mauro Mario Amato

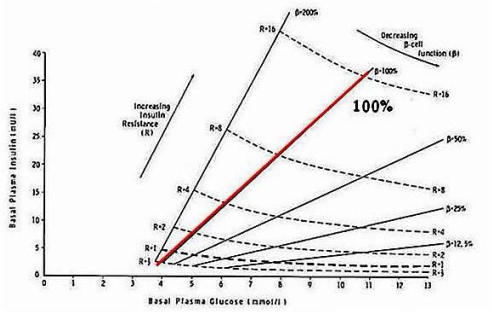
QUADERNI ENPAB

PER UNA NUOVA CULTURA PROFESSIONALE

I DATI DI LABORATORIO E LA MATEMATICA

Mauro Mario Amato

$$\text{Homa-ß} = \frac{20 \times \text{Insulinemia (mcU/ml)}}{\text{Glicemia (mmoli/l)} - 3.5} \%$$



AIP → Log (Trigliceridi / HDL-Colesterolo)

VAI (uomo) → [WC/(39.68 + 1.88 × BMI)] × (TG/1.03) × (1.31/HDL)

VAI (donna) → [WC/(36.58 + 1.86 × BMI)] × (TG/0.81) × (1.52/HDL)

- **WC** (circonferenza vita in cm)
- **BMI** (Body Mass Index in Kg/m²)
- **TG** (Trigliceridi in mmoli/l)
- **HDL** (Colesterolo-HDL in mmoli/l)

Table 1: Age-stratified cut-off points of VAI for identification of adipose tissue dysfunction (ATD).

	ATD absent	Mild ATD	Moderate ADT	Severe ADT
Age < 30 years	≤2.52	2.53–2.58	2.59–2.73	>2.73
≥30 < 42 years	≤2.23	2.24–2.53	2.54–3.12	>3.12
≥42 < 52 years	≤1.92	1.93–2.16	2.17–2.77	>2.77
≥52 < 66 years	≤1.93	1.94–2.32	2.32–3.25	>3.25
≥66 years	≤2	2.01–2.41	2.42–3.17	>3.17

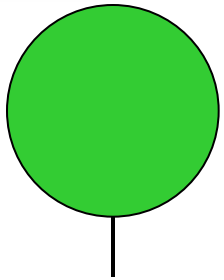
AIP = Log (Trigliceridi / HDL-Colesterolo)

InsuTAG = Insulina (mU/L) × Trigliceridi (mmoli/L)

TyG Index = Log [Trigliceridi (mg/dl) × Glicemia (mg/dl) / 2]

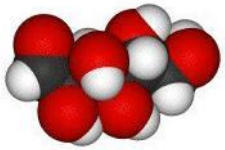
FRUTTOSAMINE

Mauro Mario Amato



NH_2

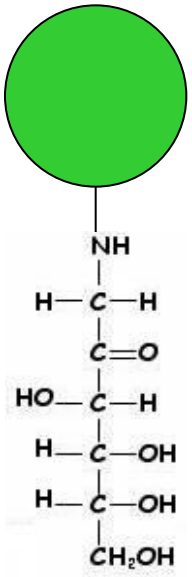
Amine
(Albumina o altre proteine)



Glucosio

esposizione delle amine a concentrazioni di glucosio più o meno elevate

glicazione



- Proteine glicate presenti in circolo in **quantità funzione del tasso glicemico**.
- Il loro dosaggio **fornisce informazioni sui tassi glicemici delle ultime 2-3 settimane** in quanto le proteine plasmatiche presentano una vita media di circa 14-21 giorni.
- **Non utilizzabile per il riconoscimento del diabete** (linee guida ADA 2019)

Fruttosamine

- **Dosaggio preferito all'HbA1c in caso di**:

  Rapidi cambiamenti nel trattamento del diabete (possibilità di valutazione della dieta o terapia in 2 settimane).

  Diabete in gravidanza (si possono osservare variazioni glicemiche significative in breve tempo).

  Perdita o anomalie dei globuli rossi (HbA1c non è più accurata)

Dosaggi: **Colorimentrico NBT**
Cromatografico HPLC

riferimenti **180-290** umoli/l

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StatPearls [Internet].

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Fructosamine

Verena Gounden; Ishwarlal Jialal.

Author Information

Last Update: January 23, 2019.

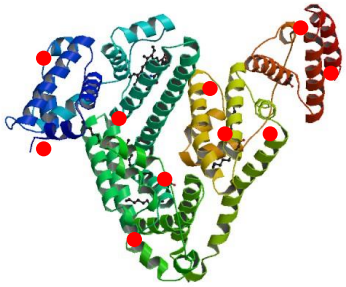
https://www.ncbi.nlm.nih.gov/books/NBK470185/

Introduction

Go to:

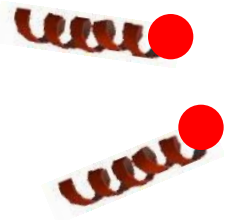
Diabetes mellitus (DM), a global epidemic, is increasing at an alarming rate and is associated with both increased morbidity and mortality. It appears that only plasma glucose and glycated hemoglobin (HbA1c) are universally accepted as reliable measures of diabetes control. Data has been evolving with other measures that bridge the span of short-term glycemic control from an isolated glucose level to HBA1c which provided an index of glycemia over 2 to 3 months. Fructosamine is a measure of non-enzymatic glycation resulting in a ketamine linkage to circulating proteins including albumin, globulins and lipoproteins and glycated albumin (GA), is a measure of the percent albumin that is glycated; both provide a measure of glycemia of 2 to 3 weeks duration. Fructosamine and GA have a potential role in the diagnoses and management of diabetes.[1][2][3]

ALBUMINA Glicata (GA)



Albumina Glicata

proteasi →



Aminoacidi Glicati

Ketoamine ossidasi →

Aminoacidi + Glucosone

H_2O+O_2 H_2O_2

H_2O_2 → POD cromogeno → **Composto colorato**

GA (Albumina Glicata)% = $\frac{\textbf{Albumina Glicata}}{\textbf{Albumina totale}} \times 100$

riferimenti → **11.9 - 15.8 %**

Caratteristiche cliniche simili alle Fruttosamine

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PMC US National Library of Medicine National Institutes of Health

PMC | Advanced | Journal list

Journal List › Med Pharm Rep › v.92(2); 2019 Apr › PMC6510364

Med Pharm Rep. 2019 Apr; 92(2): 134–138.
Published online 2019 Apr 25. doi: 10.15386/mpr-1247
PMCID: PMC6510364
PMID: 31086840

Glycated albumin is correlated with glycated hemoglobin in type 2 diabetes

Dana Mihaela Ciobanu,[✉1] Florina Bogdan,[2] Cristina-Ioana Pătruț,[3] and Gabriela Roman[1]

‣ Author information ‣ Article notes ‣ Copyright and License information Disclaimer

Abstract

Go to:

Background and aims

Glycated hemoglobin (HbA1c) retrospectively evaluates mean glycemia in the preceding 2–3 months and is the gold standard for assessing glycemic control, while glycated albumin (GA) is currently considered a short to intermediate term integrated glycemic control marker, since it reflects glycemic status over the last 3 weeks. We aimed to investigate the levels of GA, HbA1c and fasting glycemia in a group of patients with type 2 diabetes.

Methods

The observational study included adult type 2 diabetes patients (n=135) according to inclusion and exclusion criteria, randomly selected from Clinical Centre of Diabetes, Cluj-Napoca, Romania. Fasting glycemia, GA, HbA1c and creatinine were measured using commercially available methods.

Med Pharm Rep

Correlazione GA/HbA1c diabete 2

Mauro Mario Amato

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6510364/

DIABETE MELLITO GESTAZIONALE

(Intolleranza al Glucosio in Gravidanza)

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Gli ormoni placentari possono alterare il normale funzionamento dell'insulina con **aumento del tasso glicemico**.

L'aumento glicemico nel nascituro determina incremento insulinemico che, attivando anche la lipogenesi, determina aumento del grasso corporeo e quindi **incremento ponderale**.

può causare **danni al nascituro**

- **Parto distocico** o di **taglio cesareo** (percentuali di parto cesareo più alte con GDM).
- **Distress respiratorio** o **cardiaco**.

può causare **danni alla sua mamma**

- **Infezioni** ricorrenti tratto **genito-urinario**.
- Alterazioni **pressione sanguigna**.
- Aumento rischio della comparsa di **DMT2 più tardi nella vita**.
- Condizione di **pre-eclampsia** (gestosi) con edema, proteinuria e ipertensione.

Fattori di rischio

- **Età** (uguale o superiore a 35)
- **Etnia**, **Obesità**
- Storia **familiare di diabete**
- Pregressa **macrosomia fetale**
- **BMI**
  - **Basso** inferiore a **25 Kg/m²**
  - **Border-line** **25–28 Kg/m²**
  - **Alto** superiore a **28 Kg/m²**

DIABETE GESTAZIONALE (SNLG-ISS)

Raccomandazioni SID – AMD - CeVEAS

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Alla positività dell'indagine di gravidanza o al 1° appuntamento

Glicemia a digiuno >= 126 mg/dl
Glicemia random >= 200 mg/dl
HbA1c (entro 12^ sett.) >= 6.4%

Risultati confermati da un secondo prelievo → **Diabete preesistente alla gravidanza**

Gravidanza fisiologica con una delle seguenti condizioni:
- Diabete gestazionale precedente
- BMI pregravidico >= **30 kg/m²**
- Riscontro (prima o inizio gravidanza) di **glicemia tra 100 e 125 mg/dl**

OGTT 75 gr. 16-18 settimane

normale

OGTT 75 gr. 28 settimana

- Età >= 35 anni
- BMI pregravidico >= **25 Kg/m²**
- Macrosomia fetale pregressa
- Diabete gestazionale precedente
- Familiarità di diabete

OGTT 75 gr. 24-28 settimane

Glicemia (mg/dl)
a digiuno >= 92
dopo 1 ora >= 180
dopo 2 ore >= 153

Diabete Gestazionale

OGTT 75 gr. non prima di 6/8 sett. dal parto

Table 2.6—Screening for and diagnosis of GDM

One-step strategy

Perform a 75-g OGTT, with plasma glucose measurement when patient is fasting and at 1 and 2 h, at 24–28 weeks of gestation in women not previously diagnosed with diabetes.

The OGTT should be performed in the morning after an overnight fast of at least 8 h.

The diagnosis of GDM is made when any of the following plasma glucose values are met or exceeded:

- Fasting: 92 mg/dL (5.1 mmol/L)
- 1 h: 180 mg/dL (10.0 mmol/L)
- 2 h: 153 mg/dL (8.5 mmol/L)

Two-step strategy

Step 1: Perform a 50-g GLT (nonfasting), with plasma glucose measurement at 1 h, at 24–28 weeks of gestation in women not previously diagnosed with diabetes.

If the plasma glucose level measured 1 h after the load is ≥130 mg/dL, 135 mg/dL, or 140 mg/dL (7.2 mmol/L, 7.5 mmol/L, or 7.8 mmol/L, respectively), proceed to a 100-g OGTT.

Step 2: The 100-g OGTT should be performed when the patient is fasting.

The diagnosis of GDM is made if at least two* of the following four plasma glucose levels (measured fasting and 1 h, 2 h, 3 h during OGTT) are met or exceeded:

	Carpenter-Coustan (86)	or	NDDG (87)
• Fasting	95 mg/dL (5.3 mmol/L)		105 mg/dL (5.8 mmol/L)
• 1 h	180 mg/dL (10.0 mmol/L)		190 mg/dL (10.6 mmol/L)
• 2 h	155 mg/dL (8.6 mmol/L)		165 mg/dL (9.2 mmol/L)
• 3 h	140 mg/dL (7.8 mmol/L)		145 mg/dL (8.0 mmol/L)

NDDG, National Diabetes Data Group. *ACOG notes that one elevated value can be used for diagnosis (82).

ADA Diabetes 2019

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Format: Abstract Send to

Int J Gynaecol Obstet. 2019 Sep;146(3):326-332. doi: 10.1002/ijgo.12897. Epub 2019 Jul 11.

Association between glycated albumin, fructosamine, and HbA1c with neonatal outcomes in a prospective cohort of women with gestational diabetes mellitus.

Mendes N[1,2], Alves M[3,4], Andrade R[5,6], Ribeiro RT[5,6,7], Papoila AL[2,3,4], Serrano F[1,2].

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6 CEDOC Chronic Diseases - NOVA Medical School, Universidade NOVA de Lisboa, Lisbon, Portugal.
7 UA-DCM - Department of Medical Sciences, University of Aveiro, Aveiro, Portugal.

GA/Fruttosamine/HbA1c e complicazioni neonatali nel GDM

Abstract

OBJECTIVE: To investigate whether glycated albumin, fructosamine, and hemoglobin A1c (HbA1c) are associated with neonatal complications in newborns of pregnant women with gestational diabetes mellitus (GDM).

METHODS: Between November 2016 and September 2017, women with a singleton pregnancy and GDM were enrolled in a prospective study in an obstetric Portuguese referral center. Glycemic markers were compared between mothers of newborns with and without complications. Multivariable logistic regression models and corresponding areas under the receiver operating characteristic curve (AUC) were used.

RESULTS: A total of 85 women participated in the study. Raised levels of glycated albumin and fructosamine were associated with at least one neonatal complication (OR- [odds ratio] estimate: 1.33, P=0.015; OR: 1.24, P=0.027, respectively) and with respiratory disorders at birth (OR 1.41, P=0.004; OR 1.26, P=0.014, respectively). HbA1c was not associated with these outcomes. All biomarkers were associated with large-for-gestational age (LGA) status (OR 1.61, P<0.001; OR 1.45, P<0.001; OR 3.62, P=0.032 for glycated albumin, fructosamine, and HbA1c, respectively). All had similar AUC for at least one neonatal complication (0.82; 0.81; 0.79, respectively). For newborn respiratory disorders, AUCs were 0.83, 0.81, and 0.76, respectively, and for LGA status were 0.81, 0.79, and 0.71, respectively.

CONCLUSION: Raised values of glycated albumin and fructosamine were associated with particular perinatal complications in newborns of mothers with GDM, better discriminating mothers of newborns with and without complications than HbA1c.

strumenti – ottobre 2014

REGIONE AUTONOMA
FRIULI VENEZIA GIULIA

Enpab webinar

Il diabete gestazionale

Documento di indirizzo per la gestione del diabete gestazionale

Materiale Didattico

Direzione centrale salute, integrazione
sociosanitaria, politiche sociali e famiglia

Mauro Mario Amato

nutrients MDPI

Nutrients. 2019 Jul; 11(7): 1549.
Published online 2019 Jul 9. doi: 10.3390/nu11071549
PMCID: PMC6683084
PMID: 31323991

Gestational Diabetes Mellitus: The Impact of Carbohydrate Quality in Diet

Tiziana Filardi,[1] Francesca Panimolle,[1] Clara Crescioli,[2] Andrea Lenzi,[1] and Susanna Morano[1,*]

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GDM e Approccio nutrizionale LGI

Abstract

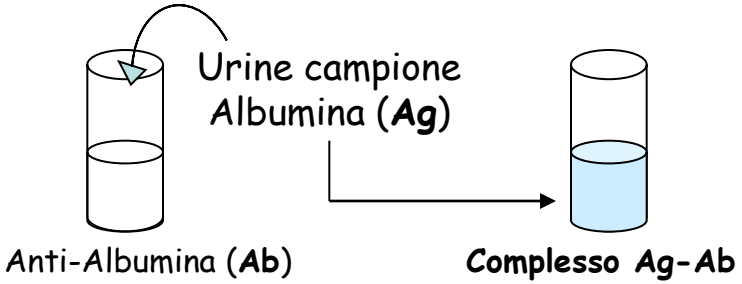
Go to:

Gestational diabetes mellitus (GDM) is defined as "glucose intolerance that is first diagnosed during pregnancy". Mothers with GDM and their infants may experience both short and long term complications. Dietary intervention is the first therapeutic strategy. If good glycaemic control is not achieved, insulin therapy is recommended. There is no consensus on which nutritional approach should be used in GDM. In the last few years, there has been growing evidence of the benefits of a low glycaemic index (LGI) diet on diabetes and cardiovascular disease. The effect of a LGI diet on GDM incidence has been investigated as well. Several studies observed a lower incidence of GDM in LGI diet arms, without adverse maternal and fetal outcomes. The main positive effect of the LGI diet was the reduction of 2-h post-prandial glucose (PPG). Several studies have also evaluated the effect of the LGI diet in GDM treatment. Overall, the LGI diet might have beneficial effects on certain outcomes, such as 2-h PPG, fasting plasma glucose and lipid profile in patients with GDM. Indeed, most studies observed a significant reduction in insulin requirement. Overall, according to current evidence, the LGI nutritional approach is safe and it might therefore be considered in clinical care for GDM.

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6683084/

MICROALBUMINURIA

Dosaggio (immunoturbidimetrico)



L'aggiunta di urina campione contenente albumina in una soluzione in cui è presente l'anti-albumina, in un preciso rapporto, provoca una **torbidità dovuta alla formazione degli immunocomplessi** (Ag-Ab) la cui intesità, letta da uno strumento dedicato, è funzione della quantità di albumina presente nell'urina campione.

- Il suo dosaggio consente di **monitorare il paziente diabetico per** il **rischio** di problemi **cardiovascolari**.
- Aumento escrezione osservata anche in **soggetti obesi**.
- Evidente associazione tra **escrezione renale di microalbuminuria e il rischio "cardio-renale"**
- Peggiore prognosi cardiovascolare con aumento della escrezione della microalbuminuria.

Escrezione urinaria di Albumina (UAE)
(urine 24h)

↓

< 30 mg/24h

Valori di riferimento
(urine mattina)

Escrezione normale	**fino a 15 mg/l**
Border-line	**15-20 mg/l**
Escrezione anomale	**> 20 mg/l**

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Mauro Mario Amato

MICROALBUMINURIA/NEFROPATIA DIABETICA/DIABESITA'

La ricerca della **microalbuminuria** è fondamentale per la diagnosi di una **nefropatia diabetica** nel suo stadio iniziale

→ Con **microalbuminuria superiore a 30 mg/l** si ha probabilità 20 volte maggiore di sviluppare una insufficienza renale contro una microalbuminuria negativa.

Microalbuminuria ↑

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SINDROME METABOLICA

- Ipertensione
- Trombosi
- Infiammazione
- LDL piccole
  HDL ↓
- Glicemia ↑
  Trigliceridi ↑
  Apo B ↑

Nefropatia

Proteine di Fase acuta (PCR)

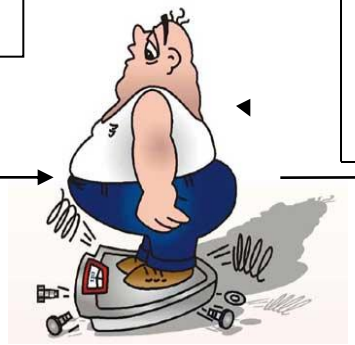
Disfunzione endoteliale

Citochine pro-infiammatorie

DIABETE

OBESITA'

DIABESITA'



Insulina ↑

Obesità viscerale

Insulino-resistenza

Adipochine/Citochine

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Molecular links between Obesity and Diabetes: "Diabesity"

Alexandra Chadt, Ph.D., Stephan Scherneck, Ph.D., Hans-Georg Joost, M.D., Ph.D, and Hadi Al-Hasani, Ph.D.

Author Information

Last Update: January 23, 2018.

Link molecolari della Diabesità

ABSTRACT

Go to:

Severe obesity represents a major risk factor for the development of type 2 diabetes mellitus (T2DM). Due to the strong association of obesity and diabetes, the term "diabesity" was coined, suggesting a causal pathophysiological link between both phenomena. The majority of individuals with T2DM are obese, highlighting the pivotal role of increased adiposity as a risk factor for diabetes. However, only a relatively small fraction of obese individuals will develop T2DM. On a population level, the link between obesity and its secondary complications is well described. However, the molecular mechanisms underlying these complications are still poorly understood. Three main hypotheses have been developed in recent years to bridge the gap between epidemiology and pathobiochemistry: (1) The "inflammation hypothesis" asserts that obesity represents a state of chronic inflammation where inflammatory molecules produced by infiltrating macrophages in adipose tissue exert pathological changes in insulin-sensitive tissues and β-cells. (2) The "lipid overflow hypothesis" predicts that obesity may result in increased ectopic lipid stores due to the limited capacity of adipose tissue to properly store fat in obese subjects. Potentially harmful lipid components and metabolites may exert cytotoxic effects on peripheral cells. (3) The "adipokine hypothesis" refers to the principal feature of white adipose cells to function as an endocrine organ, and to secrete a variety of hormones with auto- and paracrine function. Expanding fat stores can cause dysfunctional secretion of such endocrine factors, thereby resulting in metabolic impairment of insulin target tissues and eventually failure of insulin producing β-cells. For complete coverage of all related areas of Endocrinology, please visit our on-line FREE web-text,

ESAME DELLE URINE (2^ parte)

Centro Analisi Cliniche AMATO sas

Caratteristiche Chimiche

Glicosuria
Normale: assente
Reaz. Positiva:
- Iperglicemia (soglia renale 180 mg/dl)
- Insufficiente riassorbimento (disordini tubulari)
- Gravidanza (diabete mellito latente)
- Farmaci (corticosteroidi, ACTH)

Bilirubinuria
Normale: assente
Reaz. Positiva:
- Ittero ostruttivo
- Danno epatico
- Anemia emolitica

pH
Normale: 5.5 – 6.7
Urine alcaline:
- Infezioni da germi ureasi+
- Dieta
- Farmaci

Urine acide (pH<5)
- Calcolosi renale
- Acidosi diabetica
- Dieta

Chetonuria
(Acetone, Acetacetato, beta-Idrossibutirrato)
Normale: assenti
Reaz. Positiva:
- Chetosi diabetica
- Riduzione carboidrati
- Digiuno

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Sangue/Emoglobina
Normale: assente
Reaz. Positiva
- Patologie renali
- Calcoli renali
- Traumi
- Neoplasie vescica

Proteinuria
Normale: fino a 150 mg/24h
Reaz. Positiva
- Cistite e pielonefrite
- Nefropatia diabetica e CVD
- Glomerulonefrite
- Disordini tubulari e calcolosi
- LES

Urobilinogeno
Normale: fino a 0.5 mg/24h
Reaz. Positiva:
- Epatopatie ed Emolisi
- Stipsi e Sovracrescita batterica

Nitriti/Leucociti
Normale: assenti
Reaz. Positiva: Infezioni vie urinarie

Grazie per la vostra attenzione

SEE YOU SOON..!!