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Investigation into the relationship between chronic stress and insulin resistance in a Chinese population - Evidence review

Dott. A. Colonnese[1]

1 Nutritional Biologist, Italy

1. ABSTRACT

The analyzed study evaluated whether work-related stress and cortisol, a hormone produced in response to stress, were associated with insulin resistance in 766 Chinese workers aged between 40 and 60. Participants with higher job demands and greater job insecurity more frequently exhibited insulin resistance; higher cortisol concentrations were also associated with higher glycemia, waist circumference, and HOMA-IR index.The research contributes to the passage from "The Choice to Live in Health or Illness" by Dr. A. Colonnese and R. Panzironi, which states that chronically elevated cortisol can contribute to a reduced response to insulin. Independent studies confirm that an experimental excess of glucocorticoids can reduce insulin sensitivity and that markers of chronic stress are associated with higher HOMA-IR. However, a large prospective study did not find that salivary cortisol predicted new cases of insulin resistance. The most prudent conclusion is therefore that the link is moderately supported, but not proven as a sole cause in humans.

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MISSION

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2. ANALYZED STUDY

The research question was whether work-related psychosocial stress and the activation of the stress hormone system were associated with insulin resistance. The hypothesis was that working conditions perceived as more burdensome or insecure would be accompanied by higher concentrations of cortisol and lower insulin efficacy.Insulin resistance indicates that muscles, the liver, and adipose tissue respond less effectively to insulin; consequently, to keep blood glucose stable, the body tends to produce more insulin. In this study, it was estimated using HOMA-IR, an index calculated from fasting glycemia and insulin. A higher value indicates lower insulin sensitivity.The relevance to paragraph 4.14 of “The Choice to Live in Health or Illness” by Dr. A. Colonnese and R. Panzironi is precise: the publication documents the observational segment of the narrative: work stress higher cortisol higher HOMA-IR. However, it did not directly measure the activity of IRS-1, PI3K, AKT, or mTOR; therefore, it does not demonstrate the entire molecular chain described in the paragraph within the human sample.Because the design is cross-sectional, meaning it is based on measurements collected during the same period, it can identify associations but cannot establish whether stress or cortisol caused the insulin resistance. It is also possible that pre-existing metabolic conditions influence cortisol regulation or that unmeasured factors contribute to both conditions.

3. MATERIALS AND METHODS OF THE ORIGINAL STUDY

The study was observational and cross-sectional. Between March and June 2014, 766 employees from four companies in Beijing were recruited, selected from a group of thirteen companies whose workers underwent annual health check-ups. The participants were between 40 and 60 years old; 335 were men and 431 were women. The average age was 46.98 years, and 86.03% performed office work.Subjects undergoing pharmacological treatment for high glycemia or lipids, recent steroid users, and individuals with acute inflammatory diseases, liver, kidney, or heart failure, mood disorders, autoimmune diseases, or cancer were excluded. After an overnight Fasting, between 7:30 and 8:30 in the morning, glucose, insulin, cortisol, blood lipids, blood pressure, body mass index, and waist circumference were measured.Work-related stress was assessed using the short Chinese version of the Copenhagen Psychosocial Questionnaire, a 34-question survey. Higher values on the "work demands" and "job insecurity" scales indicated greater work-related tension. Insulin resistance was defined as belonging to the highest quartile of HOMA-IR, i.e., HOMA-IR equal to or greater than 2.37.The authors calculated correlations between cortisol and metabolic variables separately by sex. They then used mixed linear models, which account for the clustering of workers within the same company, to evaluate the association between cortisol and HOMA-IR after adjusting for age, smoking, alcohol, physical activity, metabolic parameters, and obesity estimated via waist circumference or body mass index.

4. RESULTS

In the overall sample, 194 people were in the insulin resistance group and 572 in the group without insulin resistance, according to the threshold adopted by the authors. Compared to the group without insulin resistance, the group with higher HOMA-IR reported greater job demands, with mean values of 58.53 versus 50.07, and greater job insecurity, 52.71 versus 39.82; both differences had $P < 0.001$. A low P-value indicates that the observed data are poorly compatible with the null hypothesis of no difference, but it does not, by itself, prove a causal relationship.The two most relevant dimensions of stress, job demands and job insecurity, were associated with both HOMA-IR and cortisol. After adjusting for cortisol, the relationship with HOMA-IR remained positive: for job demands, the partial correlation was $r = 0.149$, $P < 0.001$; for job insecurity, $r = 0.117$, $P = 0.001$. In practical terms, higher levels of these two forms of stress coincided with a higher insulin resistance index, but the strength of the correlations was modest.Dividing the participants based on the median cortisol level, the group with higher cortisol had higher mean glycemia, 5.48 versus 5.23 mmol/L, higher insulin, 8.48 versus 7.64 uIU/mL, and higher HOMA-IR, 2.08 versus 1.81; for all three differences, $P < 0.001$. Waist circumference was also greater, 81.34 versus 79.28 cm, $P = 0.005$.The correlations between cortisol and HOMA-IR were positive in both men ($P = 0.001$) and women ($P < 0.001$). Cortisol was also positively correlated with glucose,

insulin, and waist circumference in both sexes. In the model adjusted for waist circumference, cortisol remained a positive predictor of HOMA-IR in men (coefficient b = 0.003; P = 0.008) and in women (b = 0.004; P = 0.002). Replacing waist circumference with body mass index, the association remained positive in both sexes.The result directly relevant to the book is therefore this: in the sample examined, greater work-related stress was associated with higher cortisol, and higher cortisol was associated with higher HOMA-IR, even after various statistical adjustments. The study did not show that stress necessarily causes a persistent increase in cortisol in every individual, nor that cortisol is the only mediator of insulin resistance.

5. INTERPRETATION

The simplest biological explanation is that, in the individuals examined, a more intense hormonal response to stress coincided with lower insulin efficiency in controlling glucose. Cortisol normally helps the body make energy available during acute demand. If exposure is prolonged or excessive, it can promote increased glucose production by the liver and reduced glucose utilization in peripheral tissues.The result is consistent with the idea that stress can influence energy homeostasis, i.e., the balance with which the body produces, uses, and stores energy. However, the statistical association alone does not identify the mechanism. The study did not measure the phosphorylation of IRS-1, a protein that transmits the insulin signal, nor the PI3K/AKT/mTOR pathway, an intracellular network that contributes to glucose uptake and the regulation of cell growth.The segment documented by the publication is therefore observational: higher work-related stress and higher cortisol are associated with higher HOMA-IR. The next step, according to which cortisol directly alters insulin signaling in tissues, requires different experimental evidence. Controlled studies with hydrocortisone, a pharmaceutical form of cortisol, show that an increase in the glucocorticoid can effectively reduce the action of insulin on glucose utilization [1].The strength of the original work derives from the relatively large sample size, the assessment of multiple confounding factors, and the separate analysis by sex. The main limitations are a single morning measurement of cortisol, which does not describe hormonal exposure over time, the use of HOMA-IR instead of more direct dynamic measures, and the impossibility of establishing the direction of causality.

6. INDEPENDENT CONFIRMATORY EVIDENCE

Observational confirmation consistent with the book's narrative comes from a cross-sectional study conducted on 234 non-diabetic police officers, who are considered exposed to frequent work-related stress. Participants with insulin resistance had a higher median urinary cortisol compared to those without insulin resistance, 119.70 versus 89.80 ng/mg of creatinine, P < 0.001. Urinary cortisol was positively associated with HOMA-IR, with a coefficient B = 0.003 and P = 0.02 [2]. This study confirms the direction observed in the analyzed publication, but the relationship did not remain significant after adjustment for metabolic syndrome or its components. Consequently, it suggests that abdominal adiposity and metabolic alterations may be important elements of the link, rather than simple secondary variables.A second study used hair cortisol, a biomarker that can reflect cumulative exposure over weeks or months better than a single blood draw. In 527 pregnant women in Lima, higher hair cortisol concentrations in both the first and second trimesters of pregnancy were associated with higher HOMA-IR after adjustment for relevant factors. The coefficients were 0.14 (95% confidence interval 0.01 to 0.26) and 0.17 (95% confidence interval 0.03 to 0.30), respectively [3]. The result confirms, in a specific physiological context, that an indicator of chronic cortisol exposure can be associated with lower insulin sensitivity. However, it does not prove that this occurs in the same way outside of pregnancy, nor that psychological stress is the sole origin of the increase in cortisol.The most useful evidence for evaluating causal plausibility comes from a small controlled study on nine healthy subjects. After 80 mg of oral hydrocortisone, compared to placebo, the amount of glucose required to maintain stable glycemia during a hyperinsulinemic-euglycemic clamp—a technique that directly measures the response to insulin—was 35% lower, P < 0.01 [1]. This indicates that insulin was less effective in promoting glucose utilization. The suppression of hepatic glucose production was also reduced. This evidence supports the biological

pathway of elevated cortisol lower insulin sensitivity, but it concerns acute pharmacological exposure and a very small sample; it is not equivalent to demonstrating that chronic work-related stress produces an identical effect in every person.Taken together, these studies form a convergent but incomplete chain: independent observational studies associate cortisol markers with higher HOMA-IR [2,3], while a controlled experiment demonstrates that acutely increasing exposure to hydrocortisone can reduce insulin action on glucose metabolism [1].

7. CONFLICTING EVIDENCE

Significant conflicting evidence comes from the prospective population-based CoLaus/PsyCoLaus study. Among 1,525 adults, 1,149 without diabetes at baseline were followed for a median of 5.3 years. During the follow-up, 376 participants developed insulin resistance and 32 developed type 2 diabetes. Neither baseline nor evening salivary cortisol, divided into quartiles, was associated with the incidence of insulin resistance or diabetes after adjustment for age, sex, body mass index, physical activity, and smoking [4].This result does not deny that a pharmacological excess of glucocorticoids can impair insulin action. Instead, it indicates that a single assessment of salivary cortisol in generally healthy adults may not be a sufficient predictor of new cases of insulin resistance. Differences in the type of biomarker, daily cortisol variability, actual stress exposure, visceral adiposity, and the populations studied may explain part of the discrepancy.Conflicting evidence therefore requires caution: it is incorrect to consider a single cortisol measurement as individual proof of insulin resistance or as a certain indicator of future risk. The link appears to be dependent on the biological context, the duration of exposure, and the individual's metabolic vulnerability.

8. QUADRO COMPARATIVO E SINTESI DELLE EVIDENZE

The table distinguishes the publication under review from the independent research used to confirm or define the narrative of the book's chapter.

Quadro comparativo

Reference	Study type	Population or model	Main result	Direction of evidence	Strength of evidence
Original study	Cross-sectional observational	766 Chinese workers aged 40-60	Higher work stress and cortisol associated with higher HOMA-IR; cortisol positive in adjusted models.	direct confirmation	moderate
[2]	Cross-sectional observational	234 non-diabetic police officers	Higher urinary cortisol in subjects with insulin resistance and positive association with HOMA-IR; relationship attenuated after metabolic adjustments.	direct confirmation	moderate
[3]	Observational	527 pregnant women	Higher hair cortisol associated with higher HOMA-IR in the first and second trimesters of pregnancy.	indirect confirmation	moderate

[1]	Human controlled study	9 healthy subjects treated with hydrocortisone or placebo	Hydrocortisone associated with lower insulin-mediated glucose utilization and lower suppression of hepatic glucose production.	biologically consistent evidence	moderate
[4]	Prospective observational	1,525 adults from the CoLaus/PsyCoLaus cohort	Baseline or evening salivary cortisol not associated with incident insulin resistance or diabetes after 5.3 years.	discordant evidence	moderate

9. SINTESI EDITORIALE E CONCLUSIONI

CONCLUSIONS

The analyzed study shows that, in middle-aged Chinese workers, higher work-related stress and higher cortisol concentrations were associated with greater insulin resistance as estimated by HOMA-IR. This finding is consistent with a possible influence of the hormonal stress response on glucose metabolism, but it does not prove that stress is a certain or sufficient cause of insulin resistance.Independent evidence makes the picture quite well-supported at a biological level and moderately supported in humans: experimental excess of hydrocortisone can reduce insulin action, and certain biomarkers of chronic cortisol exposure are associated with higher HOMA-IR. However, inconsistent prospective results require further longitudinal studies with repeated measurements of stress, cortisol, and insulin sensitivity.

Aggiornato al 24/11/2026. Questo contenuto riflette una sintesi divulgativa delle evidenze scientifiche al momento della pubblicazione e non sostituisce il parere medico.

10. TRANSPARENCY AND SCIENTIFIC RESPONSIBILITY

Artificial Intelligence was used solely as a tool for editorial and documentary support. All content, interpretations, bibliographic citations and conclusions have been verified, reviewed and approved by the author, who assumes full scientific responsibility for them.

The manuscript was also subjected to independent peer review prior to publication.

To ensure the greatest transparency, the complete history of the article, including the dates of submission, revision, acceptance and publication, as well as information on the peer review process, is available on the dedicated Article History page.

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