Obesity and kidney disease

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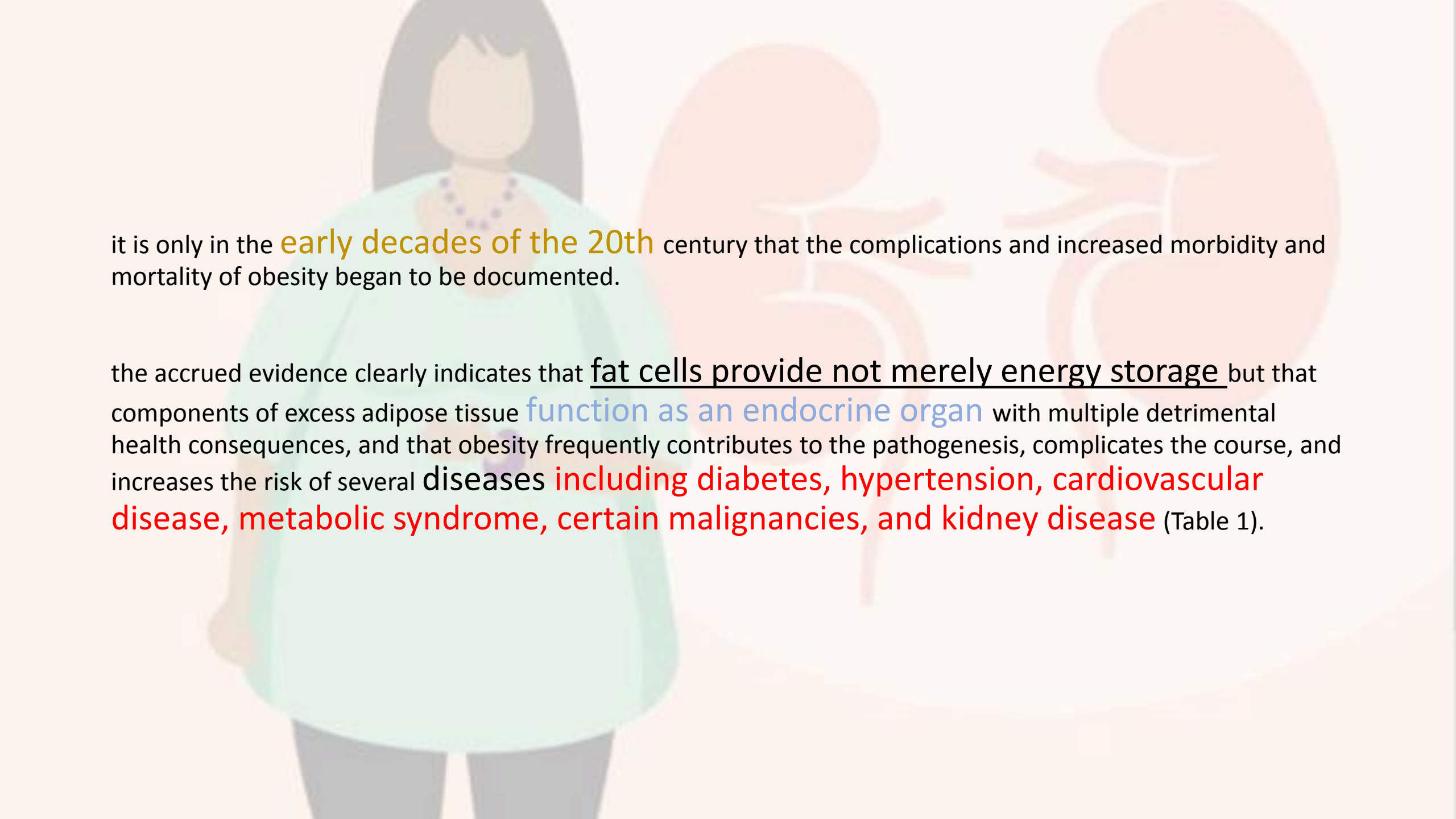

it is only in the early decades of the 20th century that the complications and increased morbidity and mortality of obesity began to be documented.

the accrued evidence clearly indicates that fat cells provide not merely energy storage but that components of excess adipose tissue function as an endocrine organ with multiple detrimental health consequences, and that obesity frequently contributes to the pathogenesis, complicates the course, and increases the risk of several diseases including diabetes, hypertension, cardiovascular disease, metabolic syndrome, certain malignancies, and kidney disease (Table 1).

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بررسی اپیدمیولوژیک عوامل جمعیت‌شناختی و پایه‌ای بالینی مبتلایان به بیماری مزمن کلیوی شهر اصفهان در بازه‌ی زمانی ۹۰-۱۳۷۵

شهرزاد شهیدی[۱]، اسماعیل هادی‌زاده[۲]، پوریا شعبانی[۲]، آوات فیضی[۳]

جدول ۱. گزارش متغیرهای جمعیت‌شناختی و پایه‌ای بالینی در بیماران

		کل نمونه	سن (سال)						جنسیت		
		۴۵۰	۴۰< ۹۵ (۱۸/۸)	۴۰-۳۰ ۹۱ (۱۸/۱)	۵۰-۴۰ ۹۳ (۱۸/۵)	۶۰-۵۰ ۹۲ (۱۸/۳)	۶۰< ۱۳۳ (۲۴/۶)	مقدار P	مرد ۲۴۵ (۴۸/۶)	زن ۲۵۹ (۵۱/۴)	مقدار P
جنسیت (مرد)		۴۸/۶	۴۲/۲	۴۶/۲	۵۰/۵	۴۶/۷	۵۳/۴	۰/۶۶۰	-	-	-
سن*		۴۶/۶۴ ± ۱۷/۰۶	۲۲/۶۵ ± ۴/۴۷	۳۳/۵۴ ± ۲/۹۲	۴۴/۱۷ ± ۲/۹۳	۵۴/۴۳ ± ۲/۵۹	۶۸/۴۹ ± ۶/۴۲	< ۰/۰۰۱	۴۷/۷۴ ± ۱۷/۴۹	۴۵/۶۰ ± ۱۶/۷۱	۰/۱۶۰
سیگار (بلی)		۴/۴	۲/۱	۴/۴	۵/۴	۴/۳	۵/۴	۰/۸۰۰	۸/۶	۰/۴	< ۰/۰۰۱
وزن*		۶۸/۰۹ ± ۱۴/۸۷	۶۱/۷۴ ± ۱۶/۴۶	۶۹/۰۸ ± ۱۲/۰۸	۷۱/۶۶ ± ۱۱/۷۷	۷۱/۴۳ ± ۱۳/۸۴	۶۷/۱۲ ± ۱۲/۹۲	< ۰/۰۰۱	۷۲/۵۱ ± ۱۳/۴۳	۶۳/۹۰ ± ۱۳/۰۸	< ۰/۰۰۱
BMI*		۲۵/۷۵ ± ۴/۸۹	۲۲/۹۵ ± ۵/۱۹	۲۶/۱۱ ± ۴/۷۴	۲۶/۶۹ ± ۴/۲۳	۲۶/۸۵ ± ۴/۶۰	۲۶/۶۹ ± ۴/۵۹	< ۰/۰۰۱	۲۵/۷۹ ± ۴/۵۱	۲۵/۷۱ ± ۵/۲۰	۰/۹۱۰
پروتئینوری	+۱	۷۹/۲	۸۴/۶	۷۹/۲	۹۲/۷	۷۴/۶	۷۰/۸	۰/۱۱۰	۷۹/۰	۷۹/۴	۰/۸۷۰

PLOS ONE

RESEARCH ARTICLE

Obesity and chronic kidney disease: A population-based study among South Koreans

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Abstract

Obesity and chronic kidney disease (CKD) are major global health problems. There are very little data concerning the prevalence and its associated factors of obesity in non-dialyzed patients who have different stages of CKD. Therefore, in this study, we examined the prevalence of obesity and its associated factors according to the stages of CKD. We used nationwide representative data from the Korean National Health and Nutrition Examination Survey, which was conducted over a 7-year period from 2008 to 2014 by the Korea Centers for Disease Control and Prevention. The results indicated that: (1) general obesity and abdominal obesity were more prevalent in patients with CKD compared to those without CKD; (2) the prevalence of general obesity and abdominal obesity was highest in stage 2 CKD; (3) stages 3a and 3b were the factors associated with general obesity, and stage 3a was significantly associated with abdominal obesity; (4) the association between general obesity/abdominal obesity and CKD disappeared in people with advanced stage 4/5 CKD; and (5) the presence of comorbidities contributed to the development of both general obesity and abdominal obesity. The findings of this study might support the idea that weight loss is a good potential intervention for the prevention of disease progression in moderate CKD (stage 3), but not severe CKD (stage 4/5).

KI REPORTS
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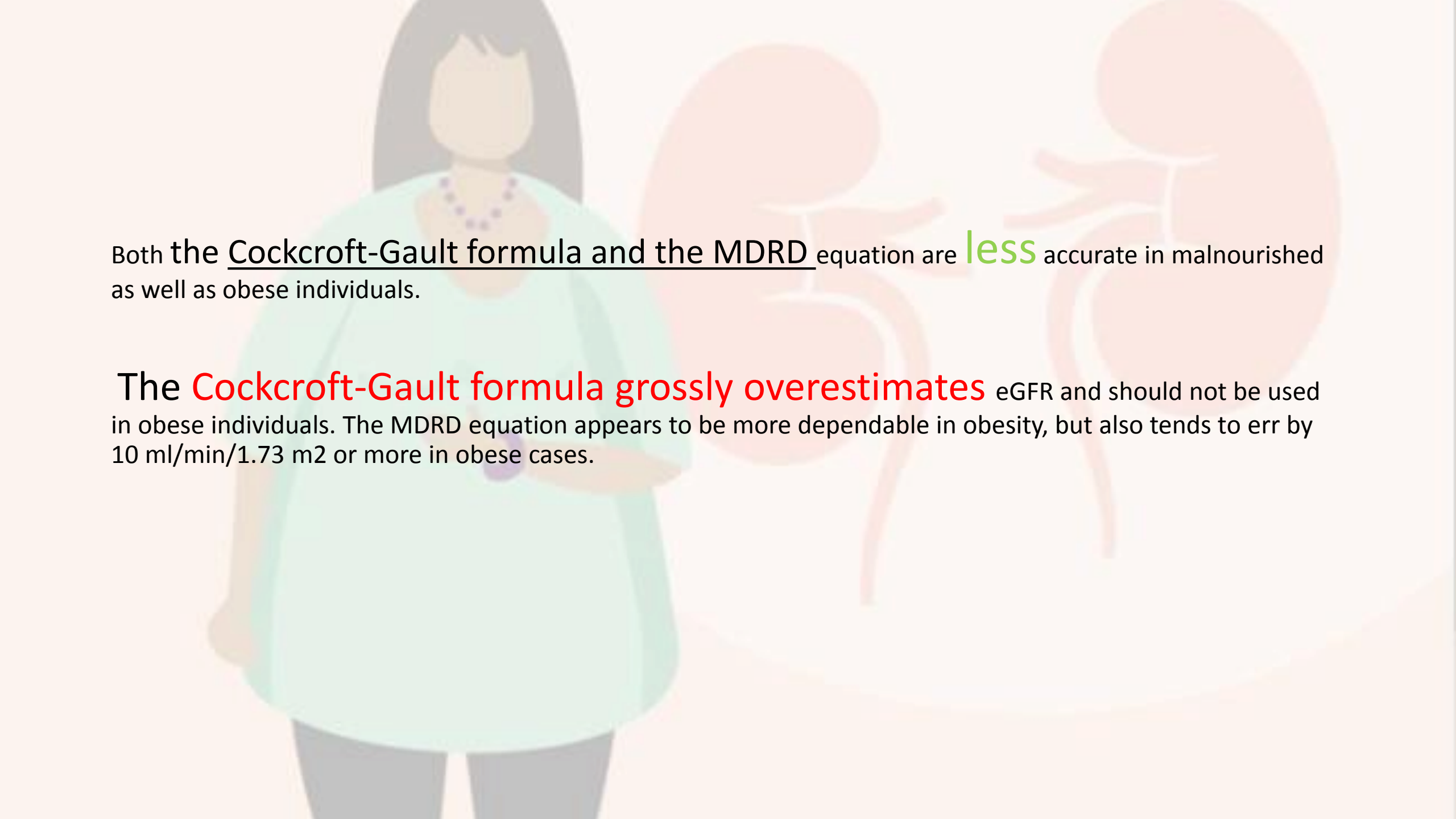
Relationship between body mass index and renal function deterioration among the Taiwanese chronic kidney disease population

Tian-Jong Chang[1,2], Cai-Mei Zheng[3,4,5], Mei-Yi Wu[3,4], Tzu-Ting Chen[6], Yun-Chun Wu[6], Yi-Lien Wu[7,8], Hsin-Ting Lin[9], Jing-Quan Zheng[1,10], Nain-Feng Chu[11,12], Yu-Me Lin[13], Sui-Lung Su[11], Kuo-Cheng Lu[14], Jin-Shuen Chen[15], Fung-Chang Sung[16], Chien-Te Lee[17], Yu Yang[18], Shang-Jyh Hwang[19], Ming-Cheng Wang[20], Yung-Ho Hsu[3,4], Hung-Yi Chiou[13], Senyeong Kao[1,11] & Yuh-Feng Lin[1,3,4,5,8,15]

This study investigated the characteristics of patients with different chronic kidney disease (CKD) stages according to various body mass index (BMI) categories and determined the influence of BMI in renal function deterioration. We conducted a multicenter, longitudinal cohort study based on the Epidemiology and Risk Factors Surveillance of CKD project (2008–2013) and National Health Insurance Research Database (2001–2013). A total of 7357 patients with CKD aged 20–85 years from 14 hospitals were included in the study. A higher male sex, diabetes mellitus (DM) and hypertension were noted among overweight and obese CKD patients, while more cancer prevalence was noted among underweight CKD patients. Charlson comorbidity index was significantly higher and correlated with BMI among late CKD patients. Patients with BMI $< 18.5\ kg/m^2$ exhibited non-significantly higher events of eGFR decline events in both early and late CKD stages than other BMI groups. BMI alone is not a determinant of CKD progression among our Taiwanese CKD patients. Obesity should be re-defined and body weight manipulation should be individualized in CKD patients.



Both the Cockcroft-Gault formula and the MDRD equation are less accurate in malnourished as well as obese individuals.

The Cockcroft-Gault formula grossly overestimates eGFR and should not be used in obese individuals. The MDRD equation appears to be more dependable in obesity, but also tends to err by 10 ml/min/1.73 m2 or more in obese cases.

PROTOCOL Open Access

Accurate GFR in obesity—protocol for a systematic review

Sriram Sriperumbuduri[1], Robert Dent[2], Janine Malcolm[3], Swapnil Hiremath[1,3], Ran Klein[4], Christine A. White[5], Pierre Antoine Brown[1,3] and Ayub Akbari[1,3*]

Discussion: This systematic review might help to inform clinicians on the best equation to use in patients with obesity and CKD for staging of CKD and for medication dosing. If no equation is deemed suitable, this review will form a basis for future studies of GFR in obese individuals.

BRIEF REVIEW www.jasn.org

Obesity in CKD—What Should Nephrologists Know?

Peter Stenvinkel,* Carmine Zoccali,† and T. Alp Ikizler‡

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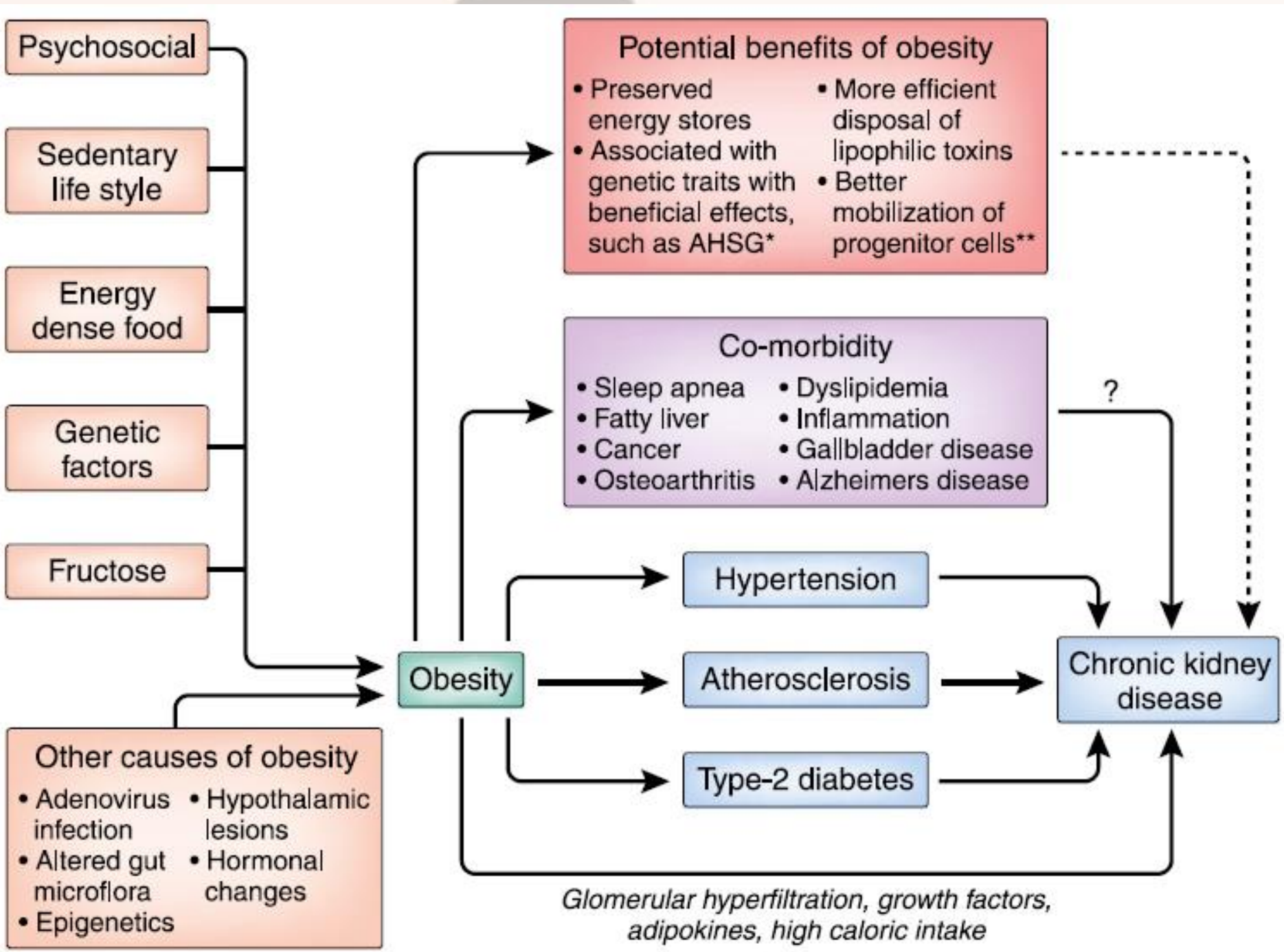
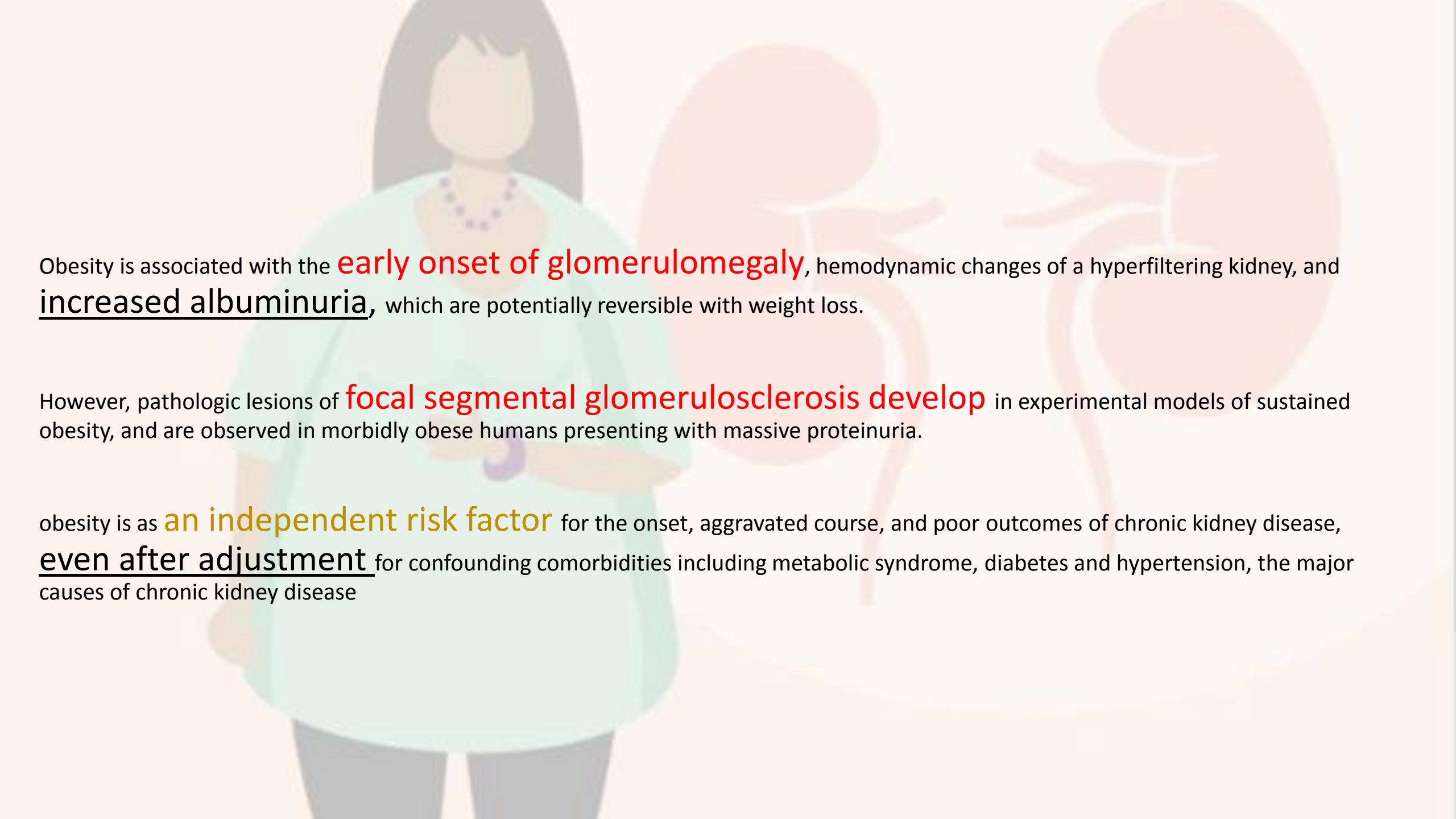


Table 1. Co-morbidities associated with overweight and obesity

1. Diabetes
2. Hypertension
3. Metabolic syndrome
4. Cardiovascular disease
5. Cancer
6. Osteoarthritis
7. Gall bladder disease
8. Non-alcoholic liver disease
9. Pancreatitis
10. Obstructive sleep apnea
11. Depression
12. Chronic kidney disease



Obesity is associated with the early onset of glomerulomegaly, hemodynamic changes of a hyperfiltering kidney, and increased albuminuria, which are potentially reversible with weight loss.

However, pathologic lesions of focal segmental glomerulosclerosis develop in experimental models of sustained obesity, and are observed in morbidly obese humans presenting with massive proteinuria.

obesity is as an independent risk factor for the onset, aggravated course, and poor outcomes of chronic kidney disease, even after adjustment for confounding comorbidities including metabolic syndrome, diabetes and hypertension, the major causes of chronic kidney disease

EFFECTS OF OBESITY ON RENAL HEMODYNAMICS

an early onset of hemodynamic changes in kidney function characterized by an increase in GFR and effective plasma blood flow, accompanied by variable increments in filtration fraction and albumin excretion in severely obese individuals

the GFR and renal plasma flow were shown to be higher by 51 and 31 percent, respectively.

a principal reason for these hemodynamic effects and attendant increase in glomerular filtration fraction is due to afferent arteriolar dilatation.

An added contributory role of efferent arteriolar vasoconstriction on the increased filtration fraction, due to stimulation of the renin-angiotensin system, has been documented also and is supported by the salutary effect of angiotensin receptor blocking drugs on the albuminuria and renal hemodynamics in experimental and clinical studies of obesity.

Evidence also exists for a role of obesity-induced increase in renal sympathetic tone that could contribute and aggravate these hemodynamic changes.

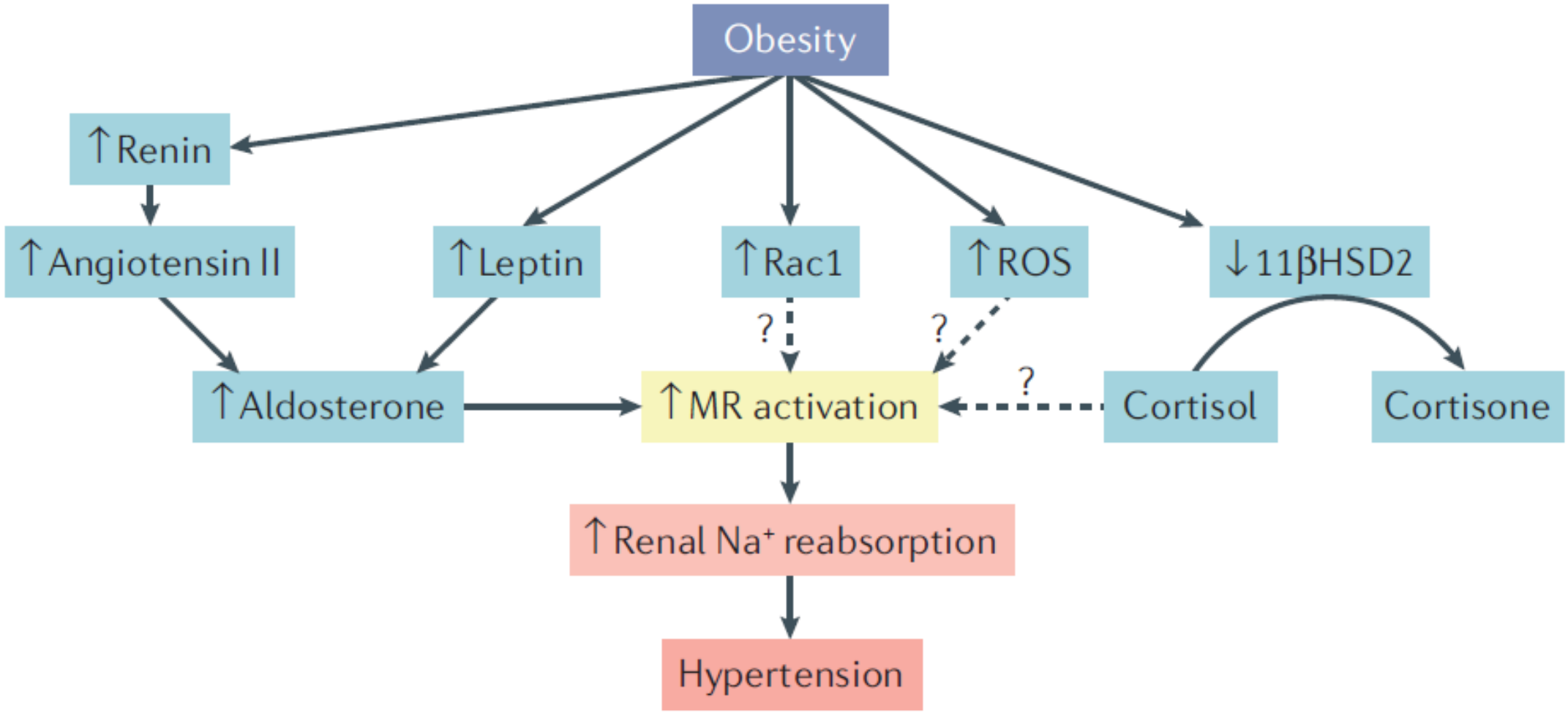
A significant correlation between increased urinary albumin excretion and body weight has been shown in both nondiabetic and diabetic overweight individuals.

impact of microalbuminuria on renal and cardiovascular risk, the prevalence of microalbuminuria (30- 300 mg/d) in men increased from 9.5% in those with normal body weight (BMI <25) to 18.3% in those who were overweight, and to 29.3% in those who were obese; in women, the respective percentages were 6.6%, 9.2%, and 16.0%.

The hemodynamic effects of overweight on kidney function and albuminuria are magnified in the presence of hypertension, which itself is a clinical complication of obesity

HYPERTENSION

Obesity, kidney dysfunction and hypertension: mechanistic links



Cytokine Role

- Leptin: adipocyte derived, structurally similar to IL2
- Crosses BBB, via reducing neuropeptide Y in hypothalamus suppresses appetite and increases energy expenditure, also incr insulin sensitivity
- Pts with obesity and metab syn are resistant to hypothalamic effects of leptin and have elvated leptin levels
- Leptin receptor Ob-Ra is expressed in kidney, and may directly affect renal structrure and function.
- Recombinant leptin stimulates prolif of cultured glomerular endothelial cells and incr TGF b1 mRNA expression and production.
- Leptin stim glucose uptake, mRNA expression TGFB type 2 rceptor, and type I collagen production in cultured mesangial cells of db/db leptin deficient obese mice. Leptin may play role in FSGS observed in obese pts with proteinuria and or ckd.

Leptin Indirect Effects

- Incr sympathetic nerve trafficking, and renal Na retention, which may cause HTN.
- Stimulates oxidative stress in endothelial clells and induces a pro-inflammatory state as a result of stim of Th1 cells. These effects may promote AS.
- Leptin shown to be an indep RF for CV events after adjustment for obesity and metab RF.
- Also, obese leptin deficient mice have been shown to be protected from AS despite presence of other RF.

IL-6 /TNF-a

- IL-6 produced from visceral and peripheral adipose cells and immune cells
- Plamsa IL-6 levels positively correlate with obesity and ins resist and predict development Type 2 DM and future coronary events.
- IL-6 shown to enhance TGF b1 signaling via modulation of TGF b1 receptor trafficking, an effect that may enhance renal fibrosis.
- TNF-a Produced by macs in adipose tissue, and levels are elevated in metabolic syndrome. TNF-a is a mediator of ins resist in adipose tissue.
- Shown to mediate inflammation in several models of renal injury, incl GN, ARF, tubulointerstitial injury. Specific role of TNF-a in metabolic syn induced renal injury has not been studied.

Adiponectin

- Insulin-sensitizing, anti-inflam, anti-atherogenic properties.
- Levels correlate negatively with fat mass, body wt, bp, insulin resistance, infalmm markers of metab syndrome.
- Low levels assoc w/vascular dysfunction and CV events
- In CKD, signif of adiponectin levels controversial

EFFECTS OF OBESITY ON RENAL MORPHOLOGY AND PATHOLOGY

the hemodynamic changes of obesity were associated with an increase in kidney weight of about 40%.

This was accompanied by an increase of glomerular size together with podocyte injury and expansion of the mesangium, and in sustained obesity resulted in mesangial sclerosis.

As with the hemodynamic changes, these early structural changes of obesity were prevented by dietary restriction

Table 2. Effects of overweight and obesity on the kidney

Hemodynamic
↑ Effective plasma flow
↑ Glomerular filtration rate
↑ Glomerular filtration fraction
↑ Albuminuria
Structural
↑ Kidney weight
↑ Glomerular planar surface
Mesangial expansion
Podocyte injury
Pathologic
Glomerulomegaly
Glomerulosclerosis
Obesity related glomerulopathy
Chronic kidney disease
↑ Onset of kidney disease
↑ Progression to kidney failure
↑ Proteinuria
End-stage renal disease
↑ Incidence and prevalence
Survival advantage in hemodialysis
↑ Graft loss in kidney transplant recipients
Other
↑ Renal cell carcinoma
↑ Nephrolithiasis

HYPERTENSION

Obesity, kidney dysfunction and hypertension: mechanistic links

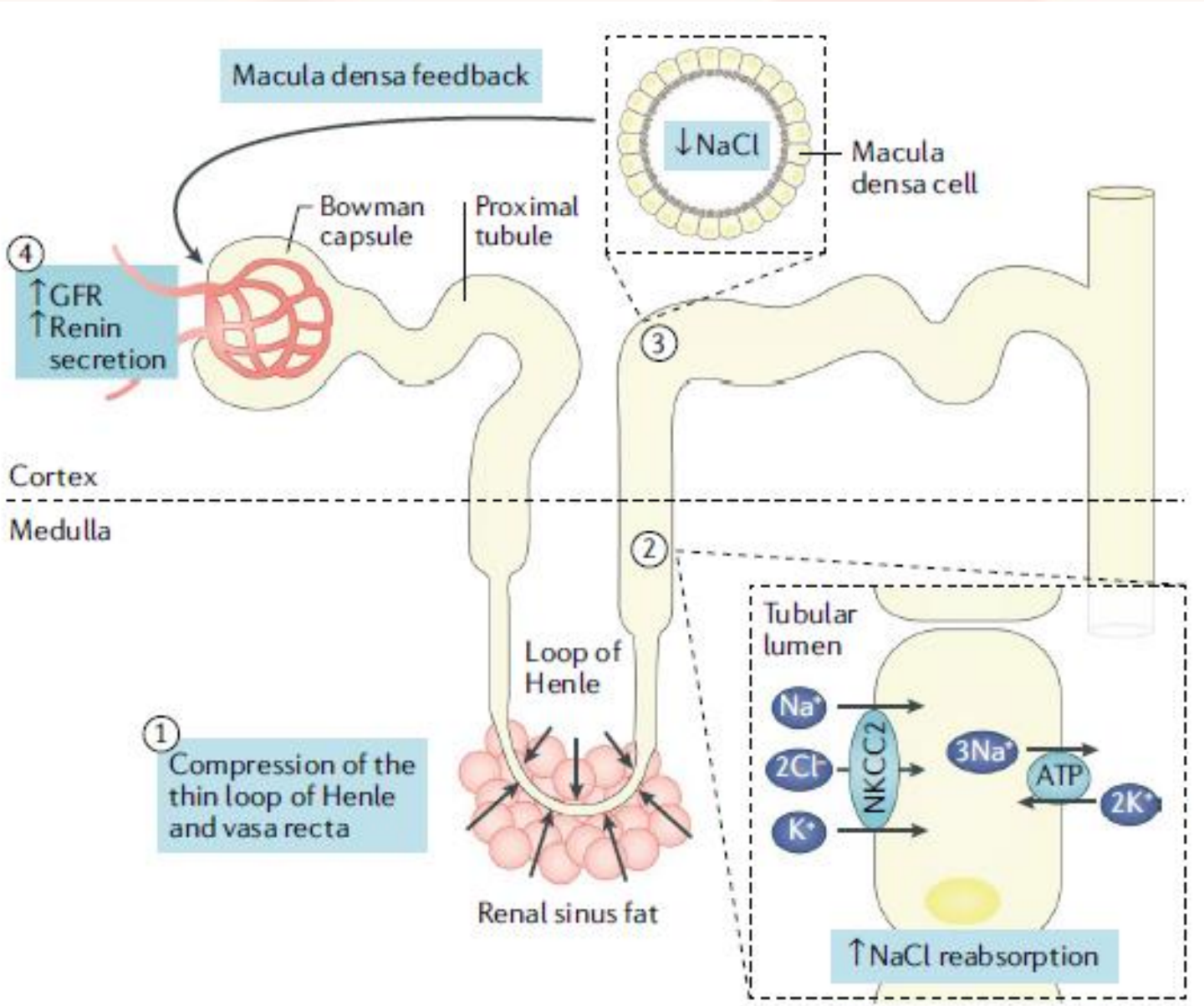
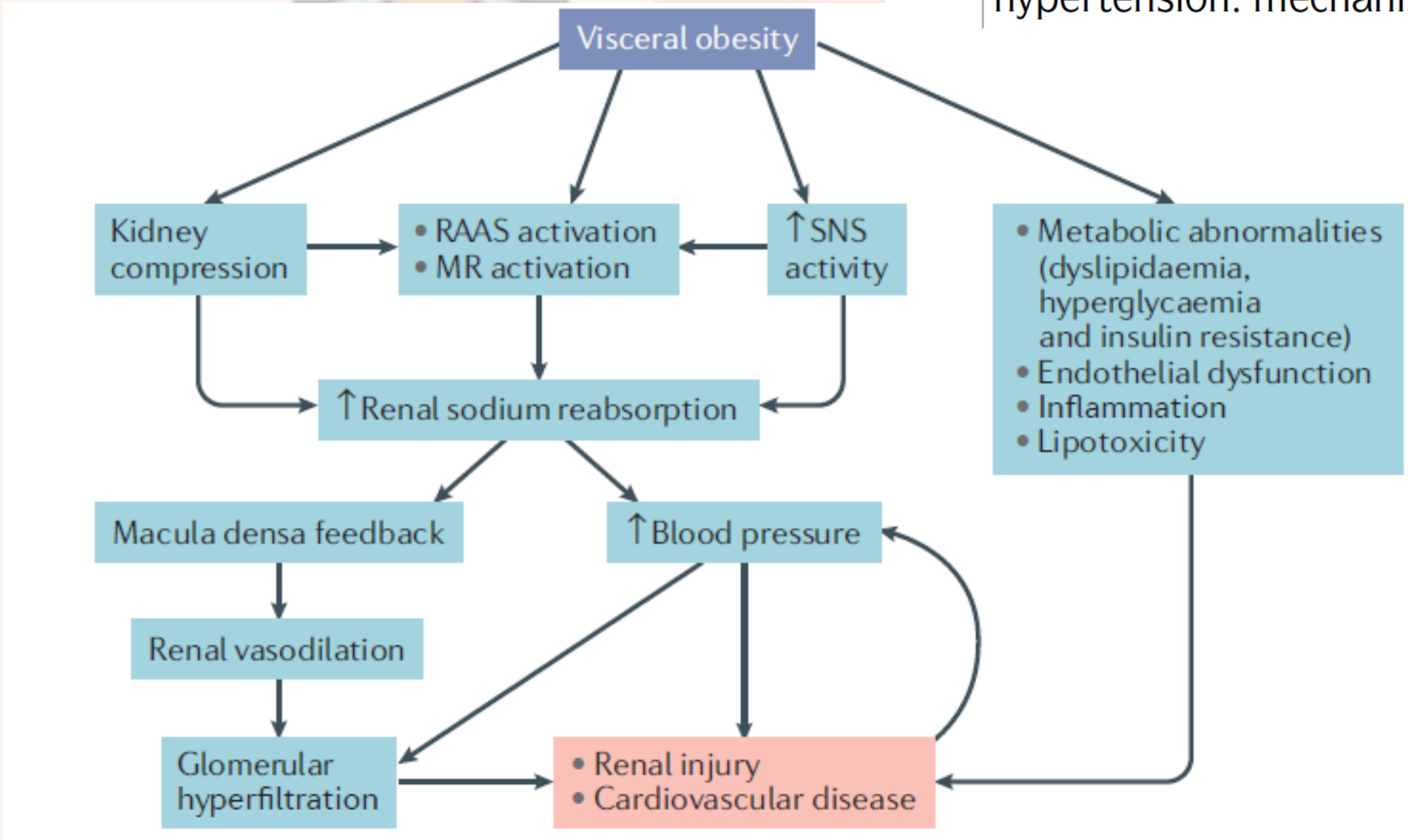
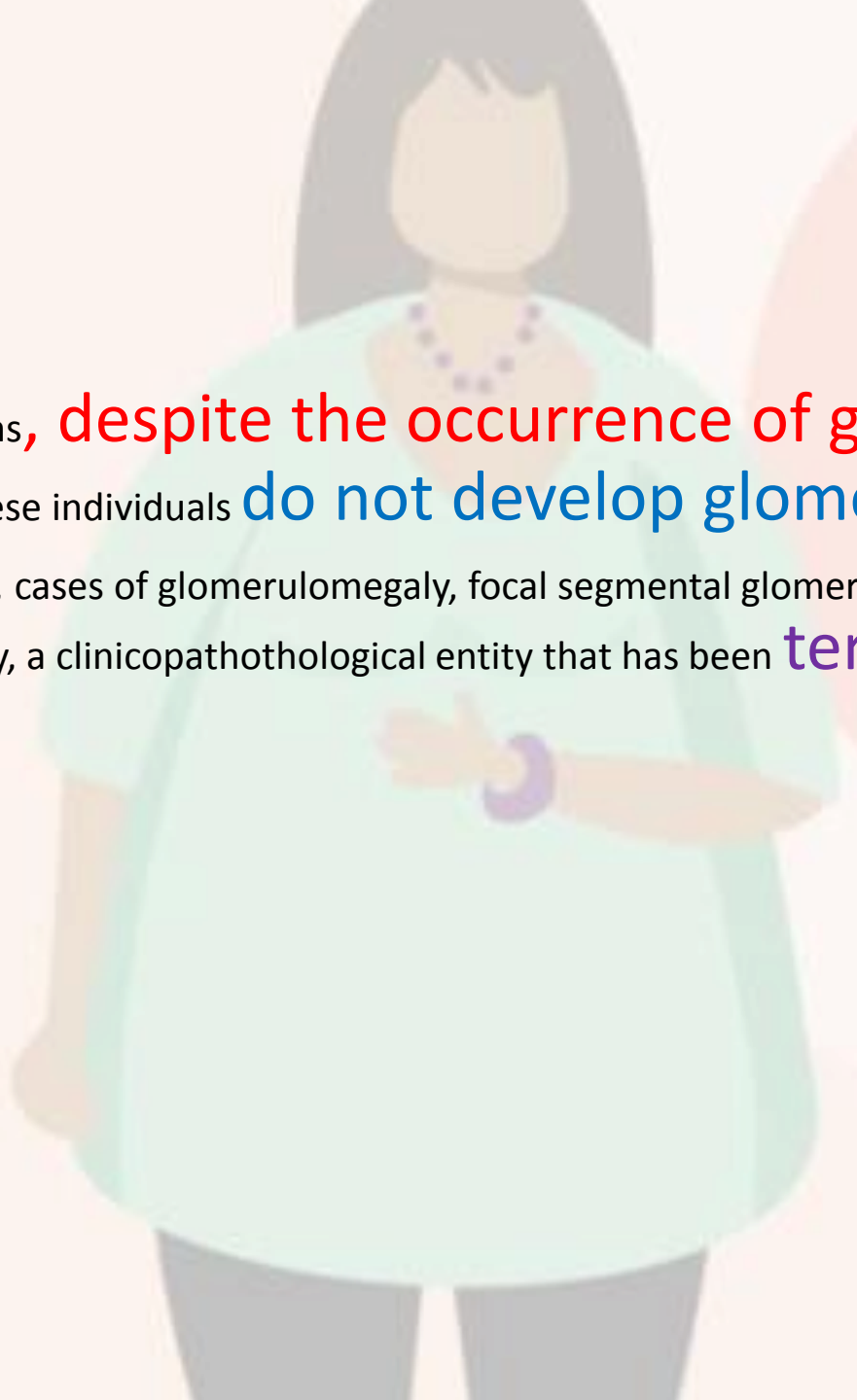


Fig. 2 | **Potential effects of kidney compression on renal haemodynamics, sodium reabsorption and renin secretion.** (1) Increased volumes of renal sinus fat and perirenal

HYPERTENSION

Obesity, kidney dysfunction and hypertension: mechanistic links





In humans, despite the occurrence of glomerulomegaly, hyperfiltration, and albuminuria most obese individuals do not develop glomerulosclerosis

However, cases of glomerulomegaly, focal segmental glomerulosclerosis, proteinuria, and decreased kidney function do occur in obesity, a clinicopathothological entity that has been termed obesity-related glomerulopathy

Obesity-related glomerulopathy: An emerging epidemic

- D'Agati, et al. Columbia Univ., KI, 2001
- 1st large renal bx-based clinicopathologic study on obesity-related glomerulopathy
- Obesity = BMI >30
- ORG-Obesity-related glomerulopathy defined as FSGS and/or glomerulomegaly
- Study to determine changing histologic incidence of ORG over past 15 yrs
- Compared cohort of ORG to controls w/idiopathic FSGS, found that there is a distinction bet these entities

Comparative Multivariate Analysis between ORG and I-FSGS

- ORG compared to I-FSGS, only parameters independently signif were serum albumin ($p<.001$) and age ($p=.032$)
- Comparing O-FSGS and I-FSGS groups, serum albumin and age were only independently signif variables.
- Results are c/w observation that major distinguishing feature between ORG and I-FSGS is the presence of full nephrotic syndrome in I-FSGS, as reflected by severity of hypoalbuminemia.

ORG = lower incidence nephrotic syndrome?

- May relate to differences in seveity of podocyte injury, in severeity and selectivity of proteinuria, and the ability of tubules ot reabsorb and catabolize the filtered protein.
- The lower fract excr B2 microglob (competes w/albumin for tubular uptake) and N-acetyl B-glucosaminidase (marker of tubular injury) obs in pts with nephrotic range proteinuria c/w those w/nephrotic syn, suggest differenc in tubular overload and resulting cellular injury.

The long term prognosis of obesity-related glomerulopathy is poor, but unlike idiopathic focal segmental sclerosis, in obesity-related glomerulopathy the incidence of nephrotic range proteinuria is lower, the serum albumin higher, the serum cholesterol lower, the edema less severe, and the progression to end-stage renal disease slower.

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The Fatty Kidney: Obesity and Renal Disease

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Table 1. Differences between obesity-related FSGS and primary-FSGS

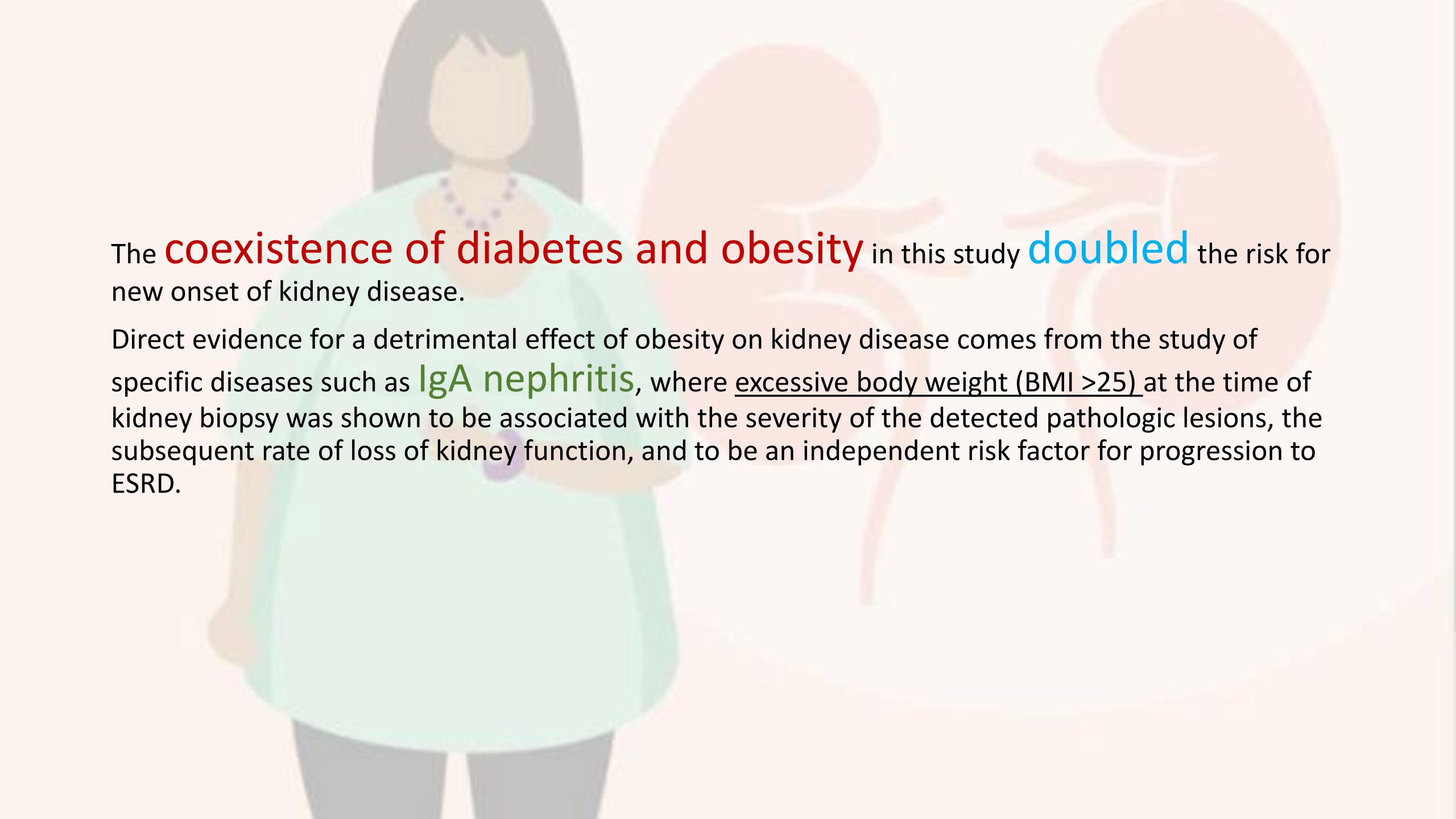
Obesity-related FSGS	Primary FSGS
Slowly increasing proteinuria	Sudden onset of proteinuria
Subnephrotic proteinuria in most of the patients	Nephrotic-range proteinuria in most of the patients
Absence of nephrotic syndrome (edema, hypoalbuminemia) even in patients with massive proteinuria	Full nephrotic syndrome frequently observed
Glomerulomegaly	Normal glomerular volume
Irregular effacement of foot processes in electron microscopy	Diffuse effacement of foot processes

EFFECT OF OBESITY ON PROGRESSION OF KIDNEY DISEASE

obesity has been shown to be associated with new onset of CKD and increased rate of progression to kidney failure in individuals with existing primary kidney disease

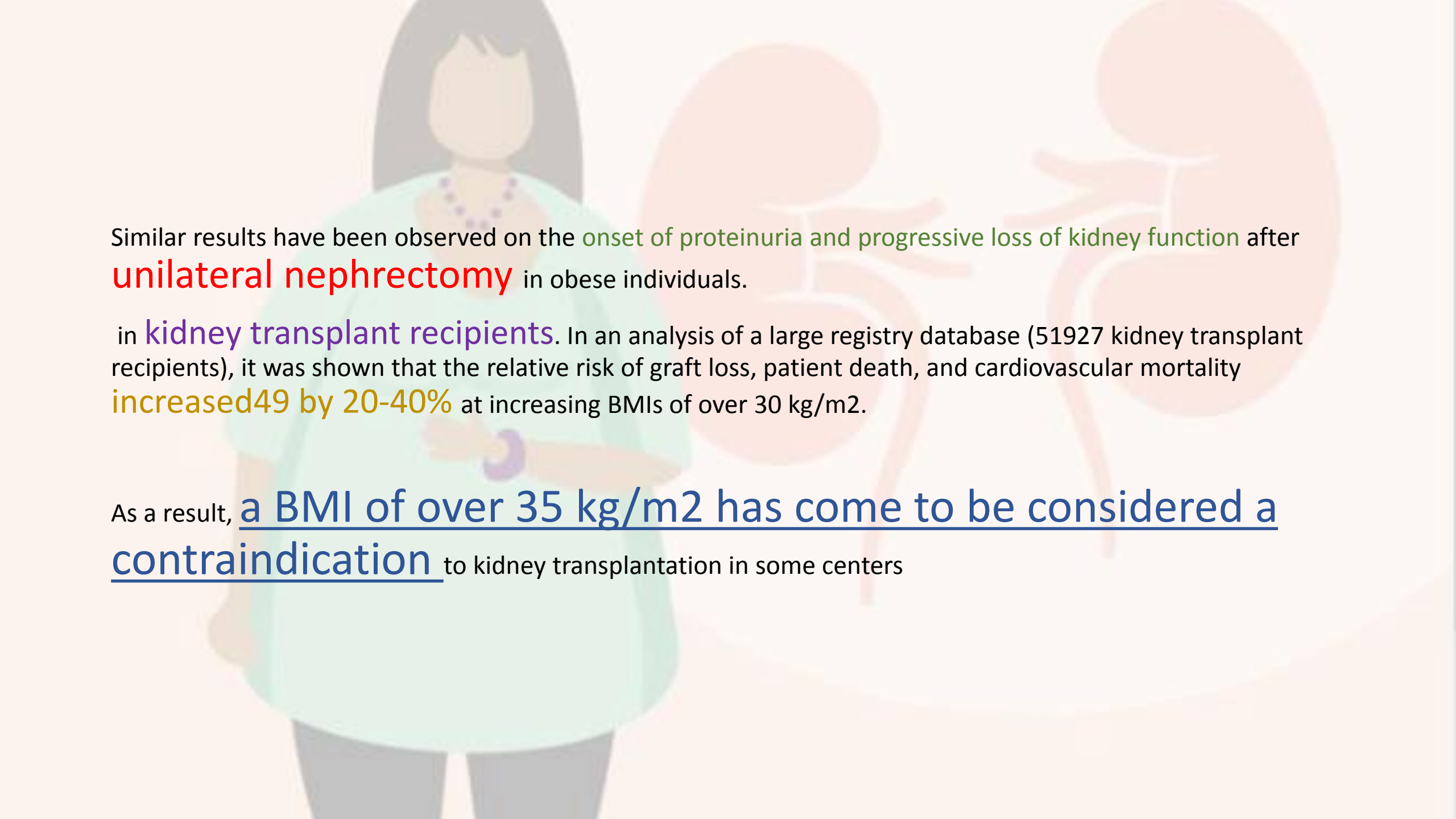
Increased BMI has also been shown to increase the risk of progression of existing kidney disease, adjusted for confounders including diabetes and hypertension.

Obese individuals with CKD have a higher rate of decline in glomerular filtration rate and progress faster to end-stage renal disease (ESRD).



The coexistence of diabetes and obesity in this study doubled the risk for new onset of kidney disease.

Direct evidence for a detrimental effect of obesity on kidney disease comes from the study of specific diseases such as IgA nephritis, where excessive body weight (BMI >25) at the time of kidney biopsy was shown to be associated with the severity of the detected pathologic lesions, the subsequent rate of loss of kidney function, and to be an independent risk factor for progression to ESRD.



Similar results have been observed on the onset of proteinuria and progressive loss of kidney function after unilateral nephrectomy in obese individuals.

in kidney transplant recipients. In an analysis of a large registry database (51927 kidney transplant recipients), it was shown that the relative risk of graft loss, patient death, and cardiovascular mortality increased49 by 20-40% at increasing BMIs of over 30 kg/m2.

As a result, a BMI of over 35 kg/m2 has come to be considered a contraindication to kidney transplantation in some centers

Increasing Body Mass Index and Obesity in the Incident ESRD Population

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J Am Soc Nephrol 17: 1453–1459, 2006. doi: 10.1681/ASN.2005111241

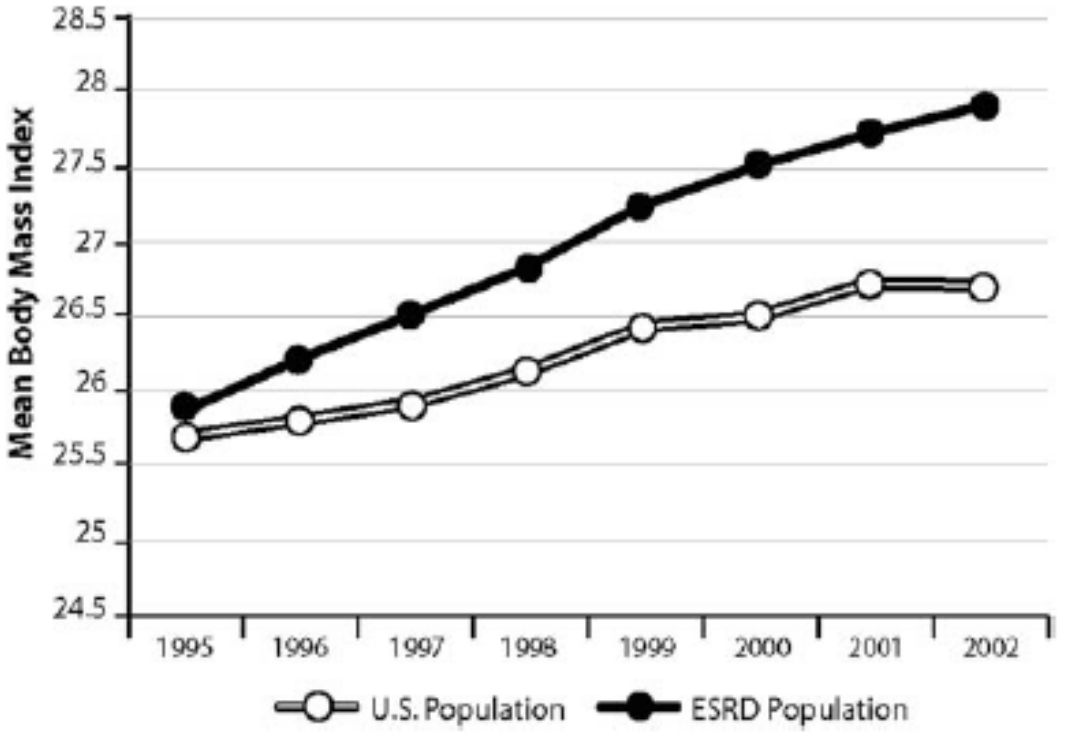

Figure 1. Temporal trends in mean body mass index (kg/m^2) among the incident adult ESRD patient population by year of first permanent dialysis initiation and in the total adult US population (Behavioral Risk Factor Surveillance System) for the corresponding year. Data are age adjusted for the 2000 US census.

PLOS ONE

RESEARCH ARTICLE

Obesity and risk of death or dialysis in younger and older patients on specialized pre-dialysis care

Ellen K. Hoogeveen[1,2]*, Kenneth J. Rothman[3], Pauline W. M. Voskamp[1], Renée de Mutsert[1], Nynke Halbesma[1,4], Friedo W. Dekker[1], for the PREPARE-2 Study Group[¶]

Method

In a multicenter Dutch cohort study, 492 incident pre-dialysis patients (>18y) were included between 2004–2011 and followed until start of dialysis, death or October 2016. We grouped patients into four categories of baseline body mass index (BMI): <20, 20–24 (reference), 25–29, and ≥30 (obesity) kg/m^2 and stratified patients into two age categories (<65y or ≥65y).

Conclusion

We found that obesity in younger pre-dialysis patients and being underweight in older pre-dialysis patients are risk factors for starting dialysis and for death, compared with those with a normal BMI.

EFFECT OF OBESITY IN CHRONIC AND ACUTE DIALYSIS PATIENTS

obesity in dialysis patients appears to provide a survival advantage, an effect that has been dubbed "reverse epidemiology".

the fact that the initial reported analysis compared variable survival data (10 years for normal, 4 years in dialyzed patients).

In a study in which subjects were followed for a comparable period, obesity was shown actually to increase mortality in dialysis patients.

KI REPORTS
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The Obesity Paradox in Kidney Disease: How to Reconcile It With Obesity Management

Kamyar Kalantar-Zadeh[1,2,3,4], Connie M. Rhee[1], Jason C
Jongha Park[4], Joline L.T. Chen[4] and Alpesh N. Amin[5]

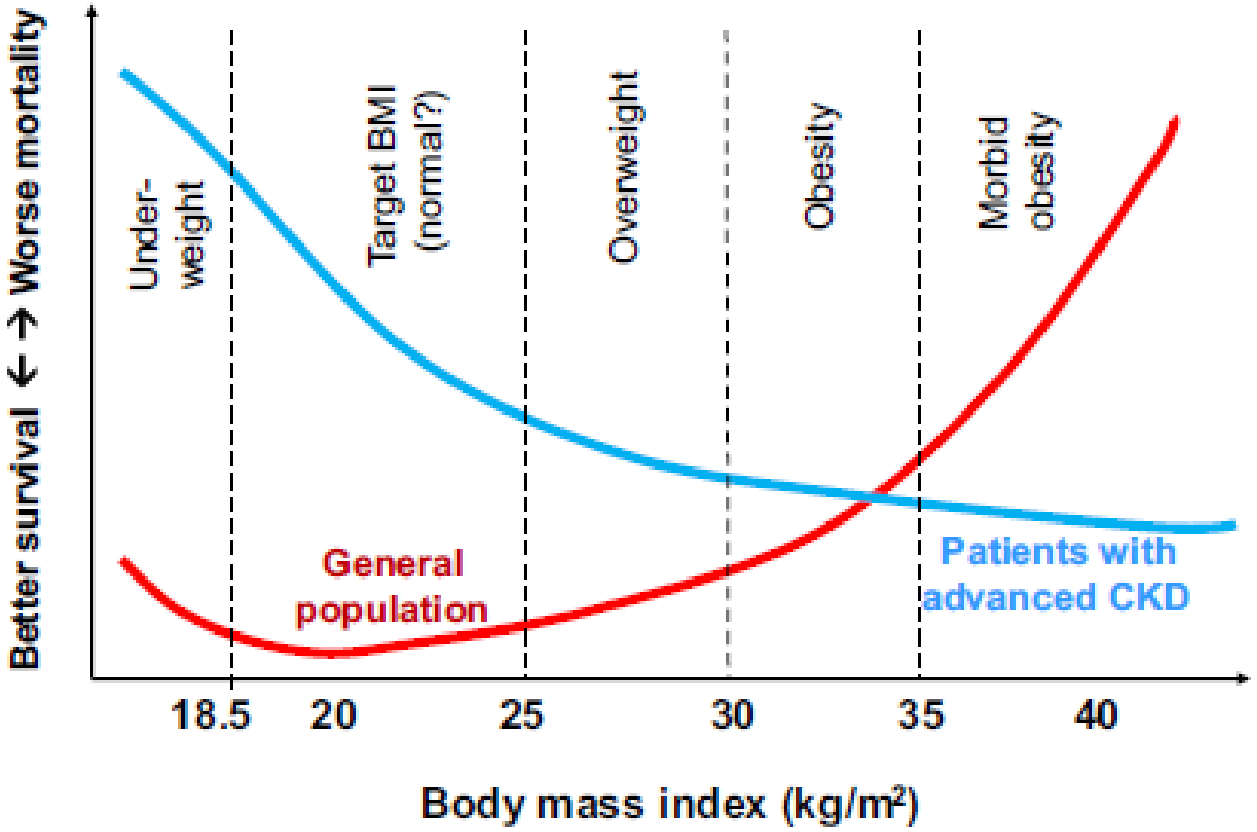

Figure 1. Reverse association of body mass index (BMI) and survival in patients with advanced chronic kidney disease (CKD) as compared to the general population.

JASN DEBATES www.jasn.org

Resolved: Being Fat Is Good for Dialysis Patients: The Godzilla Effect

ABSTRACT

Obesity is the epidemic of the 21st century. Despite the fact that obesity is known to have major health consequences in the general population, an increasing number of large-scale epidemiological studies indicate an inverse association between increasing body mass index and mortality in dialysis patients. Here it is argued pro and con that epidemiological data derived from the healthy general population may or may be not applicable to conditions such as end-stage renal disease.

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Obesity Paradox in Advanced Kidney Disease: From Bedside to the Bench.

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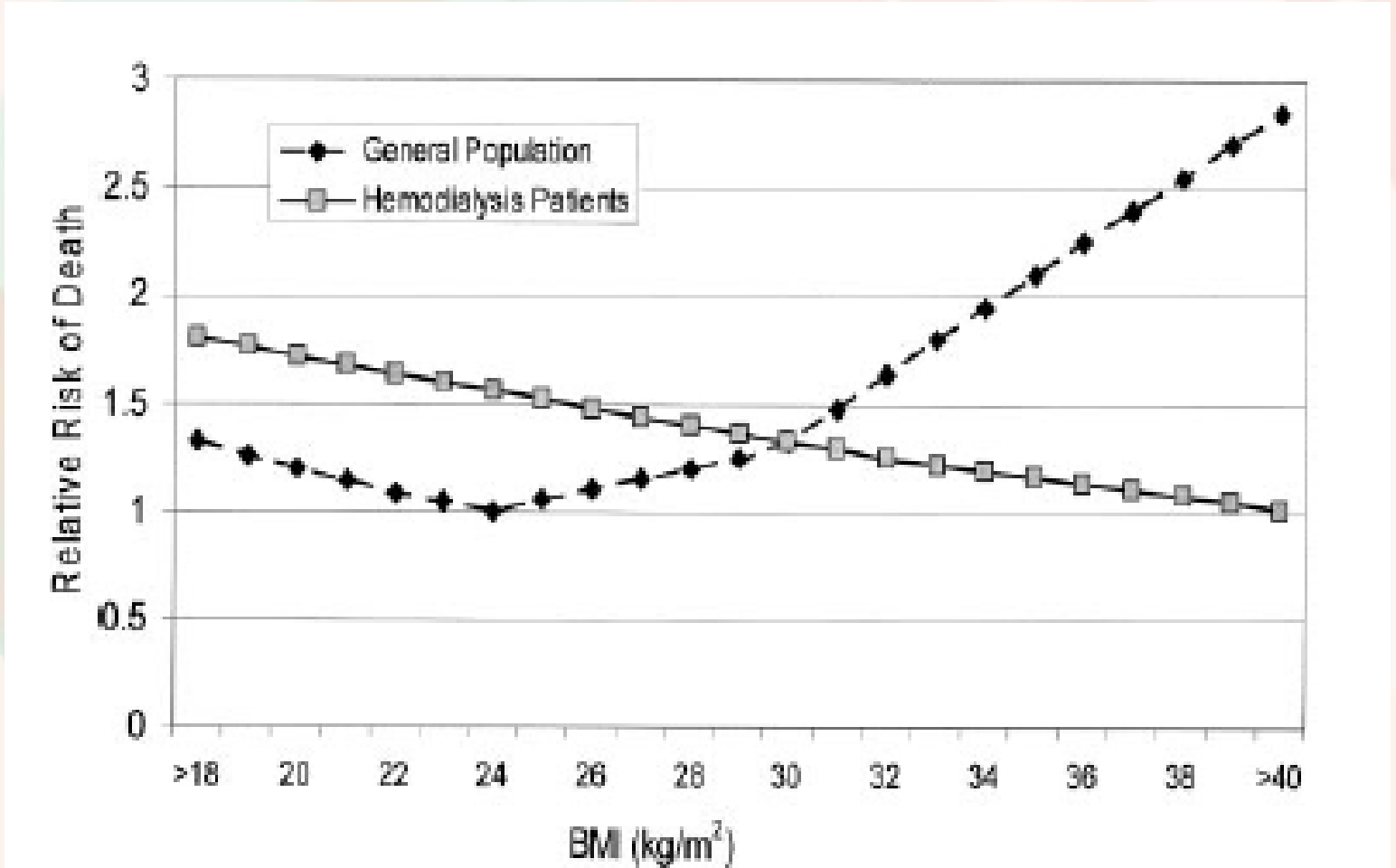
Abstract

While obesity is associated with a variety of complications including diabetes, hypertension, cardiovascular disease and premature death, observational studies have also found that obesity and increasing body mass index (BMI) can be linked with improved survival in certain patient populations, including those with conditions marked by protein-energy wasting and dysmetabolism that ultimately lead to cachexia. The latter observations have been reported in various clinical settings including end-stage renal disease (ESRD) and have been described as the "obesity paradox" or "reverse epidemiology", engendering controversy. While some have attributed the obesity paradox to residual confounding in an effort to "debunk" these observations, recent experimental discoveries provide biologically plausible mechanisms in which higher BMI can be linked to longevity in certain groups of patients. In addition, sophisticated epidemiologic methods that extensively adjusted for confounding have found that the obesity paradox remains robust in ESRD. Furthermore, novel hypotheses suggest that weight loss and cachexia can be linked to adverse outcomes including cardiomyopathy, arrhythmias, sudden death and poor outcomes. Therefore, the survival benefit observed in obese ESRD patients can at least partly be

Survival advantages of obesity in dialysis patients[1–4]

Kamyar Kalantar-Zadeh, Kevin C Abbott, Abdulla K Salahudeen, Ryan D Kilpatrick, and Tamara B Horwich



Survival advantages of obesity in dialysis patients[1–4]

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TABLE 2
Possible mechanisms leading to the observed associations between obesity and improved survival in dialysis patients

Possible mechanisms of reverse epidemiology of obesity
Malnutrition-inflammation complex syndrome (cachexia in slow motion)
Time discrepancies among competitive risk factors: overnutrition compared with undernutrition
Endotoxin-lipoprotein hypothesis
More stable hemodynamic status in obese patients
Tumor necrosis factor α receptors in obesity
Neurohormonal alterations in obesity
Reverse causation
Survival bias
Alteration of conventional risk factors in uremic milieu
Predominance of reverse epidemiology in the history of mankind

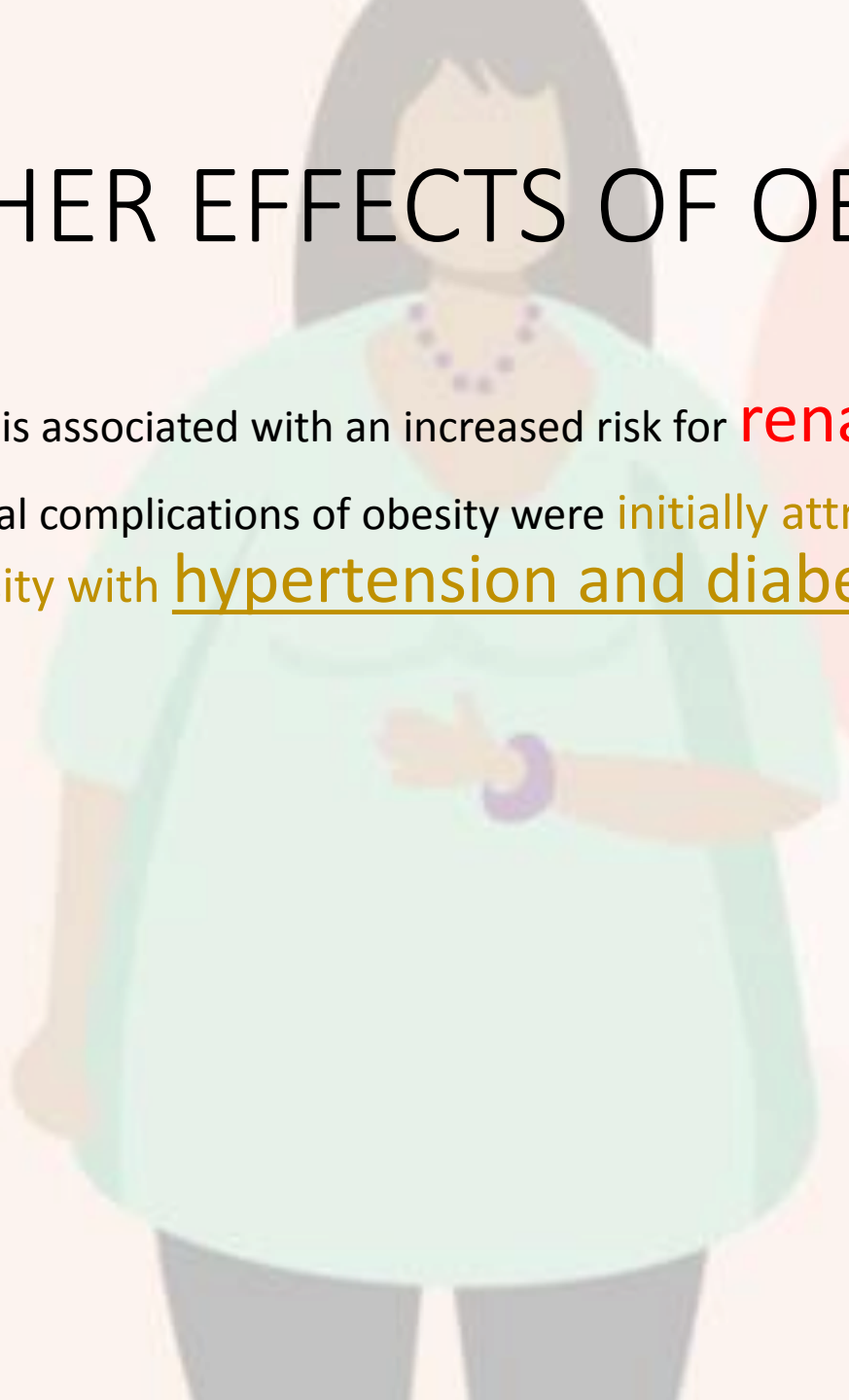
Maintenance hemodialysis is essentially a catabolic state similar to malnutrition in which obesity may convey some survival advantage to dialysis patients

The survival advantage proffered by obesity is not limited to dialyzed patients but is also observed in other chronic diseases such as congestive heart failure, liver cirrhosis, and obstructive pulmonary disease

Relevant to this issue is the finding that obesity also confers survival advantage in intensive care unit (ICU) patients requiring renal replacement therapy.

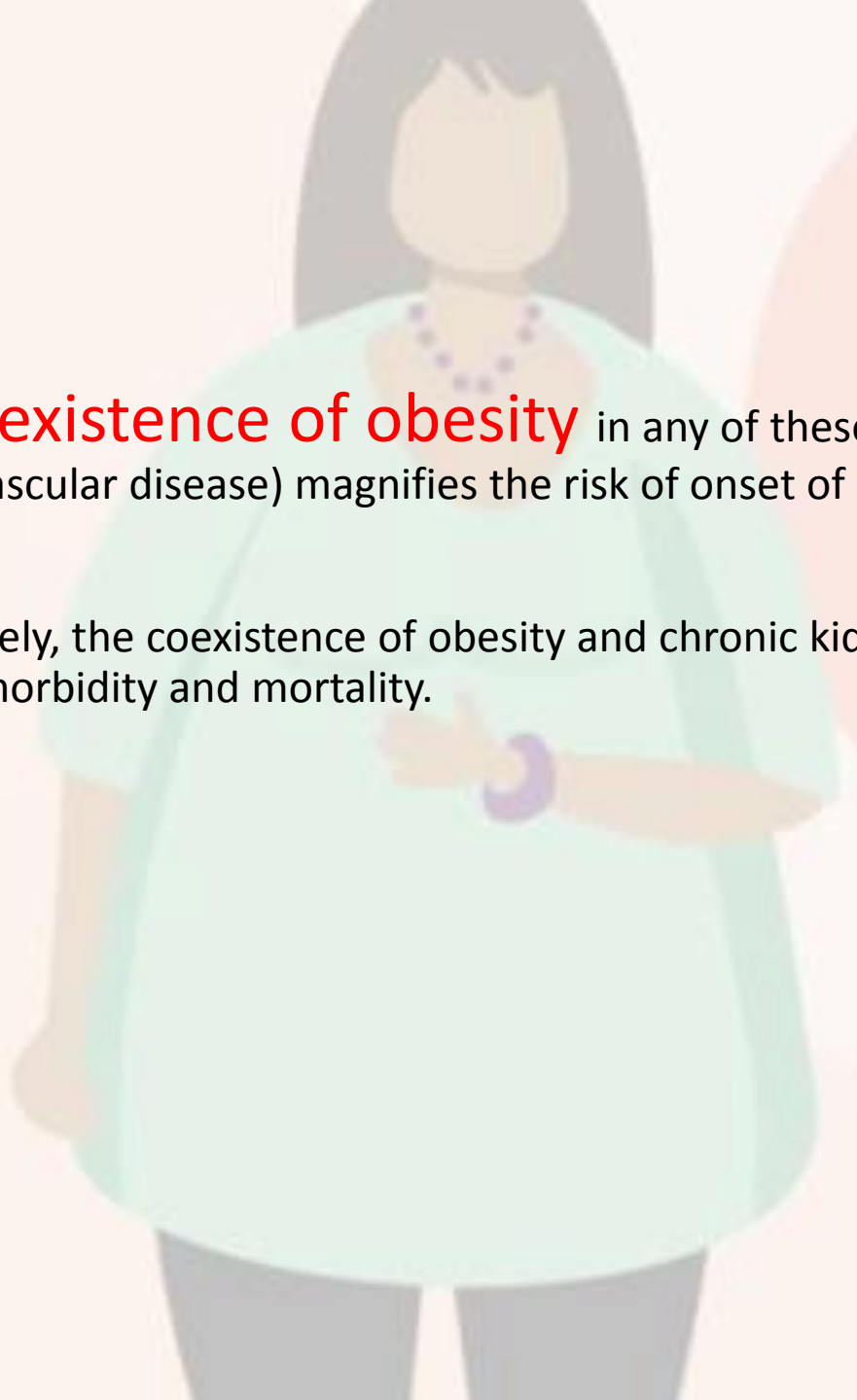
As in the case of CKD, obesity was an independent risk factor for developing acute kidney injury in the ICU, but conveyed improved survival in those requiring dialysis.

OTHER EFFECTS OF OBESITY ON THE KIDNEY



Obesity is associated with an increased risk for renal cell Carcinoma

the renal complications of obesity were initially attributed to be secondary to the common association of obesity with hypertension and diabetes, the two most common causes of chronic kidney disease



The coexistence of obesity in any of these diseases (diabetes, hypertension, metabolic syndrome, cardiovascular disease) magnifies the risk of onset of kidney disease and its progression to ESRD.

Conversely, the coexistence of obesity and chronic kidney disease in any of those other diseases increases their risk of morbidity and mortality.

HYPERTENSION

Obesity, kidney dysfunction and hypertension: mechanistic links

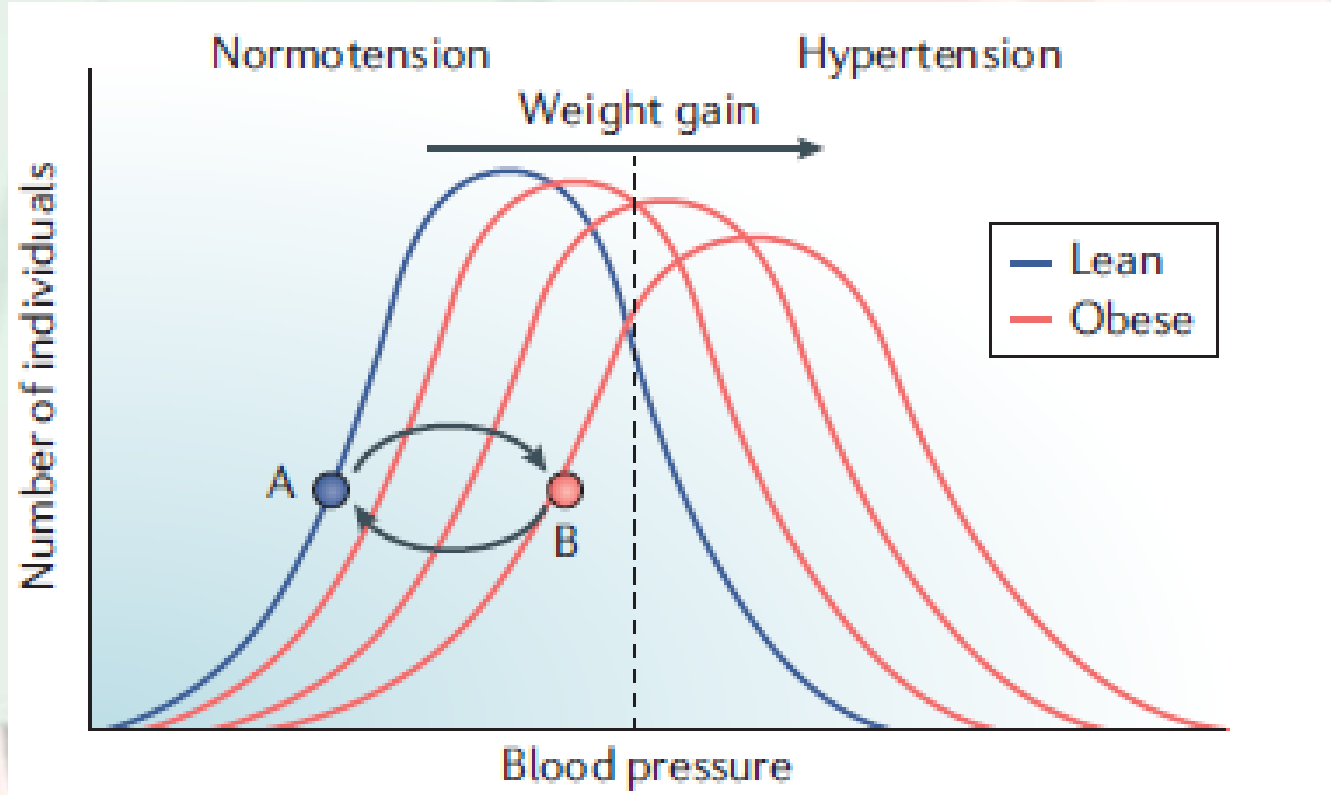


Fig. 1 | **Obesity shifts the frequency distribution of blood**

OBESITY, DIABETES, AND THE KIDNEY

Whereas thermodynamic studies clearly establish fat deposition as a consequence of imbalance between the energy derived from ingested food and that of energy expended in the course of daily activities, obesity is in fact a multifactorial disease in which the adipose tissue rather than just being a site for excess energy storage actually functions as an endocrine and exocrine organ with neurohumoral and vasoactive effects that are implicated in the genesis of obesity-related organ damage including the kidney

Treatment!

Apart from caloric restriction, given the tendency to salt retention of obesity salt restriction must be part of their dietary management.

In addition, because of the acid load of the high protein diet consumed by obese individuals consideration should be given to the use of bicarbonate supplementation in those with reduced kidney function.

Also, given the increased activity of the renin-angiotensin system in obesity angiotensin converting enzyme inhibitors and angiotensin receptor blocking agents should be part of their therapeutic regimen, especially in the presence of proteinuria and hypertension

Obesity and kidney disease

Obesidade e doença renal

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Table 1 Renal abnormalities associated with overweight, obesity and metabolic syndrome

Hemodynamic/Physiologic changes	Effective plasmatic flow increase
	GFR increase
	Filtration fraction increase
	Magnitude increase of albuminuria/proteinuria
Anatomic changes	Kidneys' weight increase
	Glomerular surface increase
	Glomerulomegaly
	Glomerular basement membrane increase
	Mesangial matrix expansion
	Mesangial cell proliferation
	Mesangial cell proliferation
	Decrease in the number of podocytes per glomeruli
	Increase in the lenght of podocyte processes
Pathology	Increase in the proportional number of glomeruli with segmental and global sclerosis
	Obesity-associated glomerulopathy/FSGS
Chronic kidney disease/ Glomerulopathies	Diabetic nephropathy
	Hypertensive neprhosclerosis
	FSGS
	IgA nephropathy
Other renal/urologic complications	Higher incidence of renal carcinoma
	Higher incidence of nephrolithiasis (uric acid and calcium oxalate)
	Higher incidence of surgical complications and graft loss in the context of kidney transplantation
End-stage renal disease (ESRD)	Higher incidence of ESRD

* GFR: Glomerular Filtration Rate; FSGS = Focal and segmental glomerulosclerosos. Adapted from Kopple & Feroze, 2011.

Thanks for attention