UNIVERSITY SIDI MOHAMMED BEN ABDELLAH
FACULTY OF MEDICINE AND PHARMACY
FEZ

Year 2016

Thesis N° 224/16

DESIGN AND DEVELOPMENT OF A MOBILE SELF-MANAGEMENT APPLICATION FOR NUTRITION IN CHRONIC KIDNEY DISEASE (CDK)

Thesis

PRESENTED AND PUBLICLY SUPPORTED THE 31/10/2016

By

MISS. GHAZZALI SALMA

BORN ON 25 JUNE 1990 IN GULMIM

TO OBTAIN THE MEDICAL DOCTORATE

KEYWORDS :

Design - Development - Self-management - Application - Chronic kidney disease

JURY

Mr. BELAHSEN MOHAMMED FAOUZI..... PRESIDENT
Professor of neurology

Mr. SQALLI HOUSSAINI TARIK ..... PROTRACTOR
Professor of nephrology

Mr. BENJELLOUN EL BACHIR……..…..... JUGE
Aggregate Professor of General Surgery

Mrs. SOUILMI FATIMA ZOHRA ..... JUGE
Aggregate Professor of pediatrics

Mr. ZARGHILI ARSALANE ……..…..... ASSOCIATE MEMBER
Information Technology (IT) professor

TABLE OF CONTENTS

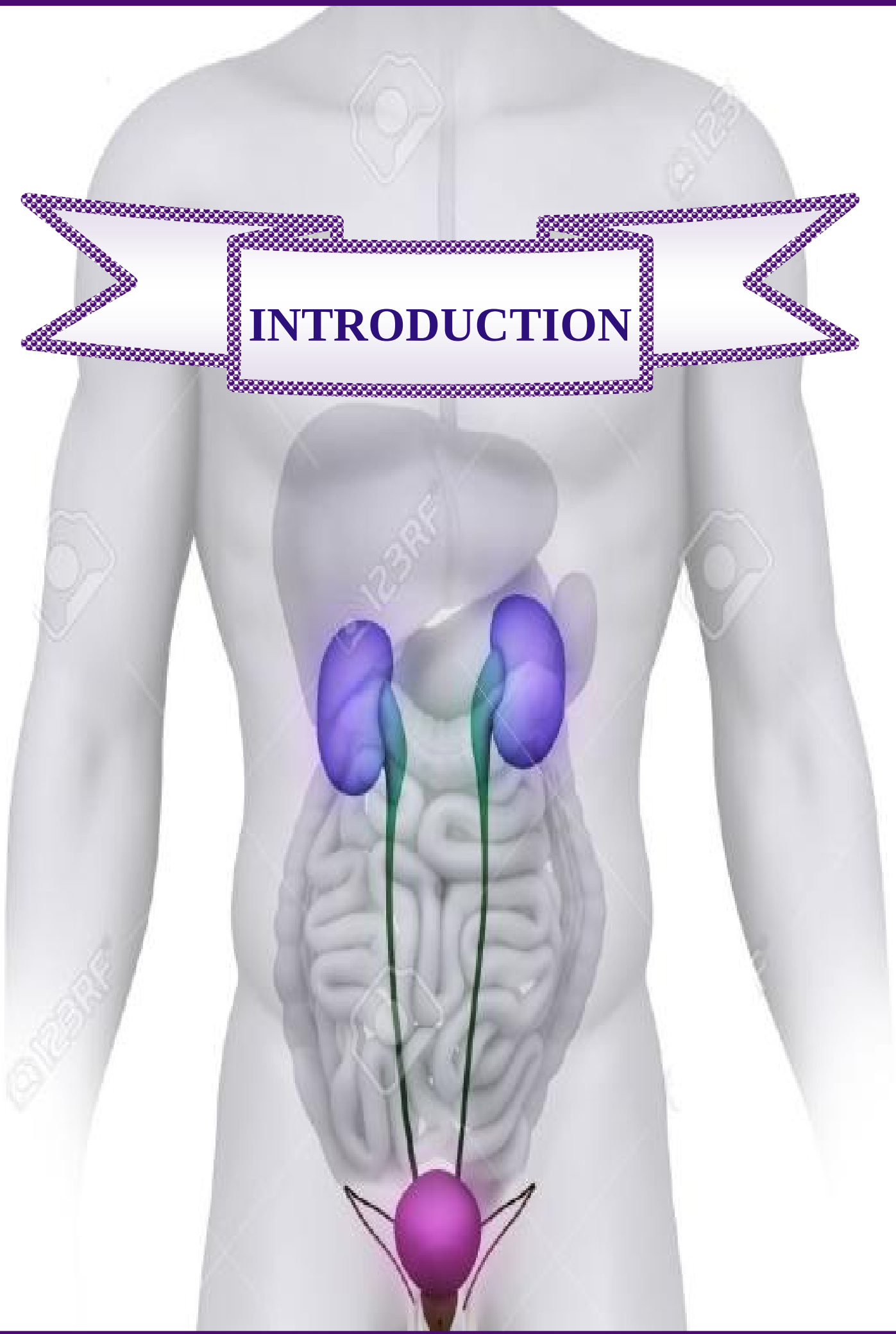
ABBREVIATIONS

AGE	: Advanced glycosylation end
ANAES	: National Agency of accreditation and health care evaluation
BNF	: British nutrition foundation
CKD	: Chronic kidney disease
CVD	: Cardiovascular diseases
DPI	: Dietary protein intake
DRI	: Dietary recommended intake
ECF	: Expansion in extracellular fluid
ESKD	: End stage kidney disease
FAO	: Food and Agriculture organization
FSD	: Functional Specification Document
FSGS	: Focal segmental glomerulosclerosis
GFR	: Glomerular filtration rate
GLUT	: Trans membrane glucose transporters
HBP	: High blood pressure
HD	: Hemodialysis
IGF-1	: Insulin-like growth factor-1
NKF-KDOQI	: National kidney foundation
PD	: Peritoneal dialysis
PEM	: Poor-Energy malnutrition
PEW	: Protein-energy wasting
PKD	: Polycystic kidney disease
RAAS	: Renin angiotensin aldosterone system
RRT	: Renal replacement therapy
SNGFR	: single nephron GFR
TFG-β	: Transforming growth factor beta
VEGF	: vascular endothelial growth factor
WHO	: World health organization

LIST OF FIGURES

LIST OF TABLES :

INTRODUCTION



1. Background

There is no doubt that chronic kidney disease (CKD),also known as chronic kidney failure or insufficiency -often leading to a gradual and irreparable loss of renal function- is a serious clinical condition that is considered as a worldwide public health issue, in view of the increasing incidence and prevalence, poor outcomes, and high cost. In fact, until a year ago, 10% of the global world population was affected [(1)]. And it is even taken as an epidemic in developed nations, notably Australia and the United states[(2)(3)] where more than 20 millions of people are concerned[(4)].This social-scale-problem, imposes tremendous clinical and psychosocial burdens, and begets fatal outcomes which include not only kidney failure but also complications of decreased kidney function and cardiovascular disease. Current evidence suggests that some of these adverse outcomes can be prevented or delayed by early detection and treatment. Unfortunately, chronic kidney disease is undertreated, particularly regarding the nutritional part of management[(5)].

2. Motivation

Patients with chronic kidney failure present a variety of metabolic and nutritional abnormalities[(7)(8)(9)], and are particularly more vulnerable to malnutrition, that arises from both pathophysiological (e.g. uremic toxicity, altered metabolism) and iatrogenic (e.g. polypharmacy, the prescription of a low protein diets to slow disease progression, disabilities and disease-related social issues) causes. With the start of dialysis, some of these abnormalities are improved, but others remain or worsen[(10)].Therefore, compromised nutritional status as a consequence of the disease is considered to be one of the major treatment priorities for patients with

established CKD, indeed dietary interventions, can slow down the progression of the disease, alleviate symptoms[11] and may also ameliorate disease complications such as hyperkalemia, acidosis and sarcopenia[12]. However, within such a chronic care, enhancing or maintaining an appropriate nutritional status is not an easy target to reach. In fact, as CKD progresses, disorders and symptoms accumulate and the demands on the patient's self-management increase, which affects patients outcomes and daily life quality. At the same time the patient's beliefs about his/her illness, treatment and own control are considered to influence each other and the patient's health behavior[13][14].

Hence, considering the primordial role of nutrition therapy in prevention and management Of CKD, and in view of the above-mentioned difficulties and constraints, faced by both patients and healthcare providers when managing the daily nutritional intakes of patients, there is a constant need for new better specific tools to improve the individual ability to manage his/her dietary requirements, their agreement on and adherence to treatment prescriptions. In reality, many applications and services have been developed to feel that need, but none of them is actually adapted to our Moroccan diet. A starting point for this thesis was the creation of a Moroccan version of such applications, within a professional perspective of patient education, and an interest in self-management when living with CKD.

3. Contributions

The major contribution of this paper is that we build a personal dietary guide application, which is usable on Android system and integrates 3G mobile phones and smart home technologies, in order to facilitate the dietary management in Moroccan CKD patients. The application has a rich database containing all the

current and personalized dietary guidelines in matters of CKD, for both adults and children, and offers an option to maintain the daily nutritional intakes in the tolerated borders, without, however, limiting the patient's personal choices, and while leaving a free space for other specific recommendations, that could be advisable by nephrologists. The data provided on the application is suited to Moroccan special dishes and eating habits. The application, not only assists with the tasks of diet management, but also improves the medical observance, gives an educational support, and helps patients to deal with their chronic condition by taking full advantage of features in existing subsystems in smart home and related cutting edge technologies.

4. Aims of the thesis :

4.1 Overall objective:

The overall purpose of this dissertation is to design and develop a mobile self-management application for nutrition management in chronic kidney disease, to use on Android operating system, in smart home devices. Which goes with the current worldwide trends, since more than one third of about 1.4 billion smart phone users in the world, have at least one medical application on their devices[15].

4.2 Specific objectives:

Several sub-objectives have motivated the project:

4.2.1 For patients with CKD:

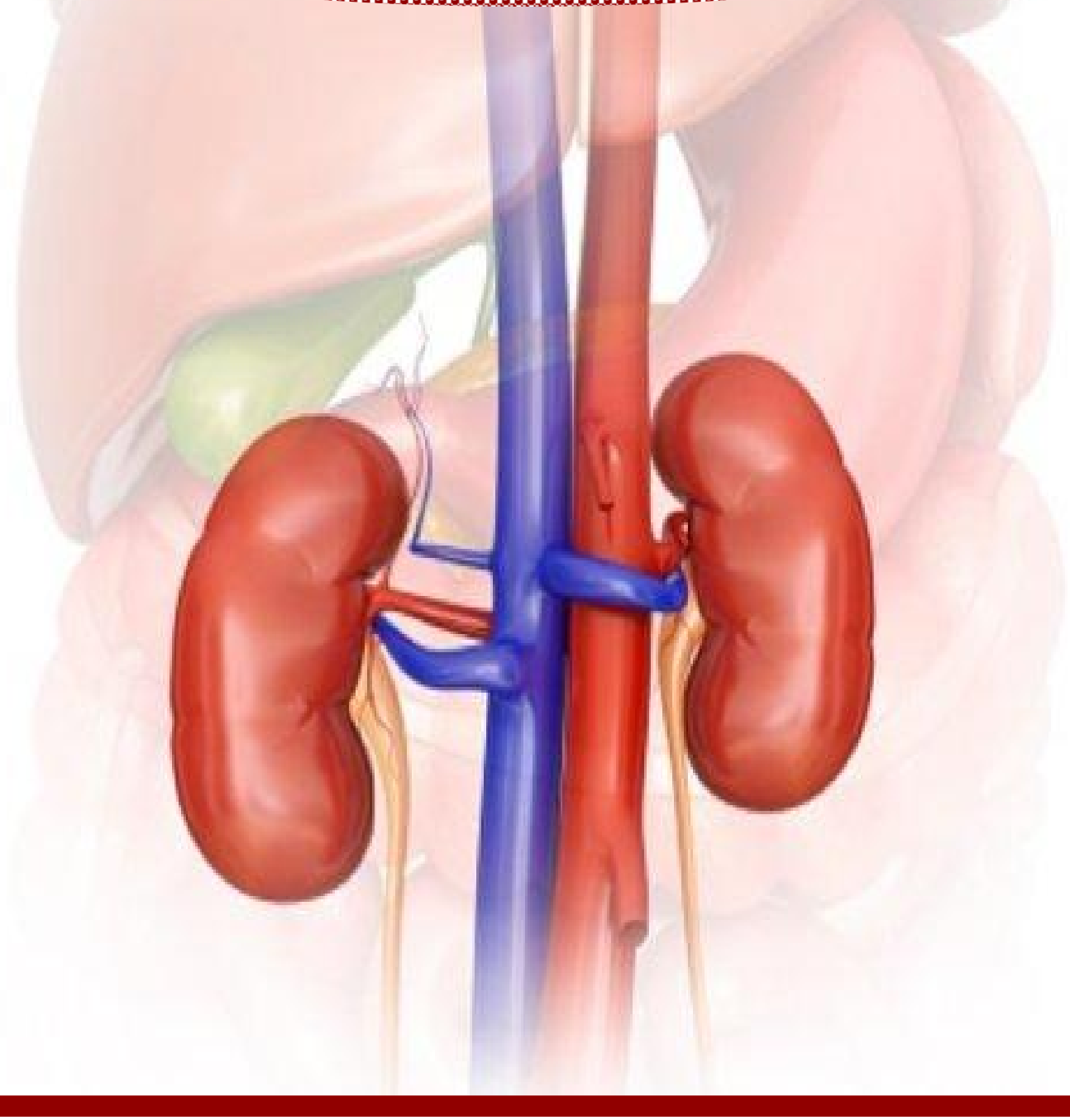
1. To assist both affected adults and children, to self-manage their nutritional status, easily, and keep away the need for patients' family members, friends and communities to involvement in the care activities.

2. Helping them to control their daily nutrients input and avoid excessive intakes.
3. Allowing them to monitor how their nutritional status evolves, over the previous days.
4. Giving them the opportunity to make free food choices, while respecting their nutritional requirements.
5. Optimizing their daily food targets and avoid malnutrition.
6. Enhancing their treatment adherence and medical compliance.
7. Improve their education level in matter of CKD.
8. Instructing them how to deal with their disease and ameliorating their life quality.

4.2.2 For the healthcare providers:

9. To facilitate the provision of healthcare, for Doctors & dietitians; with less effort and less time.
10. To improve the safety and maximize the quality of care giving by providing timely health information to professional healthcare providers.
11. To reduce the increasing burdens, challenges and shrinking professional care giving force.

LITERATURE REVIEW



The kidneys

The urinary system of the human body consists of two kidneys, two ureters, the bladder and a single urethra (figure1).The kidneys are located retroperitonieally inside the abdomen, and serve several essential regulatory roles, the most important is being; the waste filtering and disposal system of the body[16].

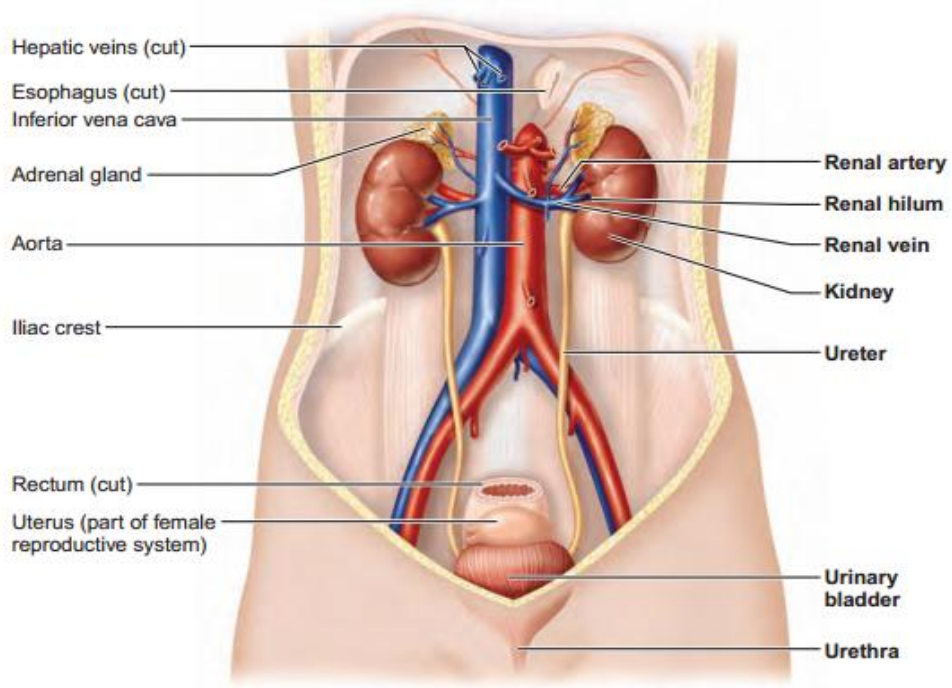


Figure1: The urinary system. Anterior view of the female urinary organs[17]. (Most unrelated abdominal organs have been omitted.)

1. Anatomy of the kidney

1.1 External Anatomy:

1.1.1 Location:

The kidneys are a pair of organs that lie in a retroperitoneal position (between the posterior muscular wall of the abdominal cavity and the parietal peritoneum), in the superior lumbar region (Figure 2a). they Extend approximately from T_{12} to L_3 , and receive some protection from the lower part of the rib cage (Figure 2b). The right kidney is crowded by the liver and lies slightly lower than the left one(17).

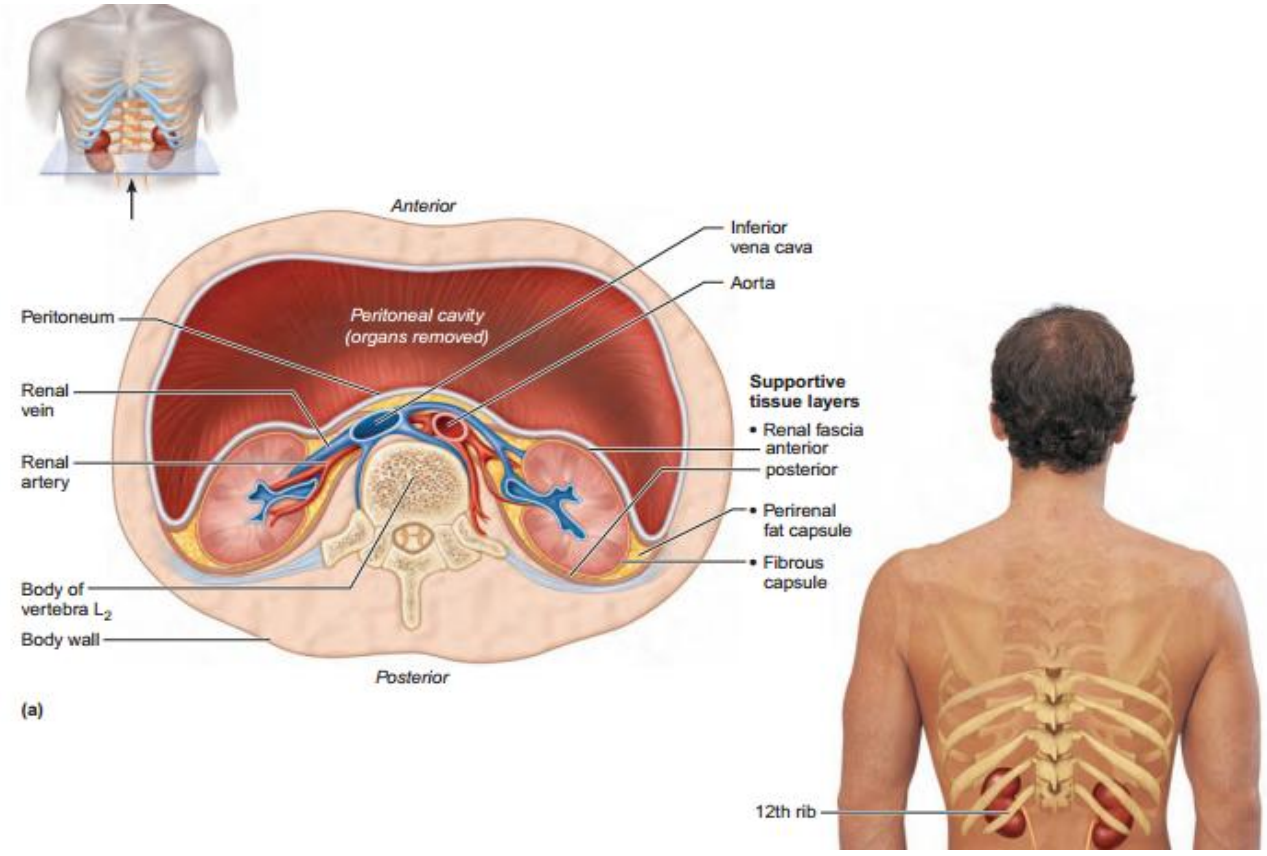


Figure 2: Position of the kidneys against the posterior body wall. (a)Cross-section viewed from inferior direction. Note the retroperitoneal position and the supportive tissue layers of the kidney. (b)Posterior in situ view showing relationship of the kidneys to the 12th rib pair (17).

1.1.2 Description:

Grossly, the kidneys are bean-shaped structures with a lateral convex surface and a medial concave surface that has a vertical cleft called the renal hilum, which leads into an internal space within the kidney called the renal sinus, where the ureter, renal blood vessels, lymphatics, and nerves all join each others, in each kidney.(17) A thin layer of fibrous connective tissue forms the renal capsule surrounding each kidney. This renal capsule provides a stiff outer shell to maintain the shape of the soft inner tissues.(18)The kidney weigh about 150 g in the male and about 135 g in the female. They are typically 10-12 cm in length, 5-7 cm in width, and 2-3 cm in thickness.(19)(Figure 3)

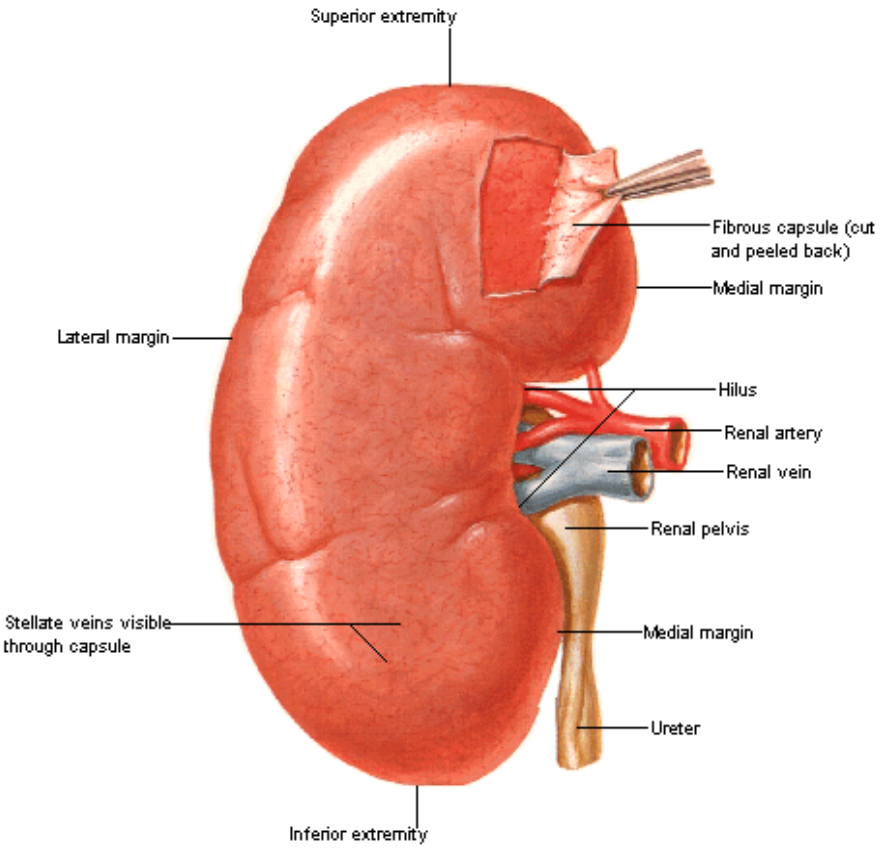


Figure 3: Gross Anatomy of the kidney(20)

1.2 Internal Anatomy:

ü macroscopic structure:

Viewed internally, The kidney is divided into the cortex and medulla. Seven cone-shaped tissue masses, called renal pyramids, form the medullary area and are separated by the cortical tissue called renal columns (of Bertin). The renal pyramids are aligned with their bases facing outward toward the renal cortex and their apexes point inward toward the center of the kidney. Each apex connects to a minor calyx, a small hollow tube that collects urine. The minor calyces merge to form 3 larger major calyces, which further merge to form the hollow renal pelvis at the center of the kidney. The renal pelvis exits the kidney at the renal hilus, where urine drains into the ureter[18].(Figure 4)

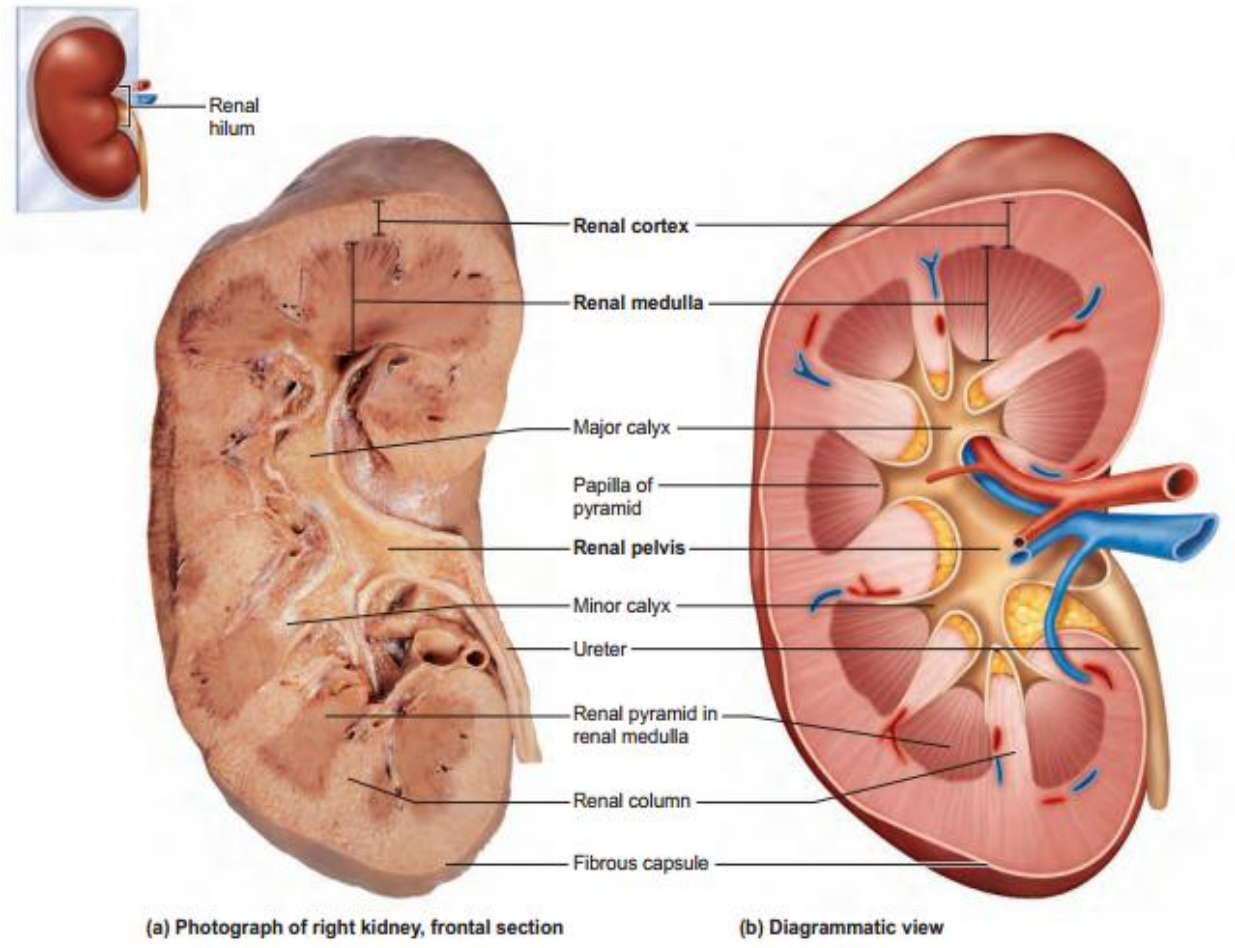


Figure4 : Internal anatomy of the kidney. Frontal sections [17].

ü microscopic structure:

The renal medullary pyramids contain microscopic coiled tubes called nephrons(figure 5); the kidneys's microscopic functional units that filter blood to produce urine. Each kidney contains around 1 million individual nephrons. Each nephron is made of 3 main parts:

Ø the renal corpuscle(Figures 5, 6);responsible for filtering the blood. It is formed by the capillaries of the glomerulus surrounded by the glomerular capsule (also known as Bowman's capsule).The glomerulus is a bundled network of capillaries that increases the surface area of blood in contact the blood vessel walls, the vascular network is formed by an afferent arteriole and an efferent. While the glomerular capsule, is a cup-shaped double layer of simple squamous epithelium with a hollow space between the layers. It presents special epithelium cells known as podocytes and which form a thin filter to separate urine from blood passing through the glomerulus.

Ø The renal tubule(Figures 5, 6);a series of tubes that concentrate urine, recover non-waste solutes from it, and carry it from the glomerular capsule to the renal pelvis. The renal tubule presents the following sections:

- A curvy first section, known as the proximal convoluted tubule, whose cells reabsorb much of the water and nutrients initially filtered into the urine.
- The loop of Henle, a long straight tubule that carries urine into the renal medulla before making a hairpin turn and returning to the renal cortex
- Following the loop of Henle is the distal convoluted tubule.
- The collecting duct, which carries the concentrated urine through the renal medulla and into the renal pelvis[18].

Ø The juxtaglomerular apparatus; Located next to the glomerulus. It is found between where blood enters a renal corpuscle and the distal convoluted tubule

of the same nephron. This location is critical to its function in regulating renal blood flow and glomerular filtration rate, The juxtaglomerular apparatus consists of three cell types : the macula densa, a part of the distal convoluted tubule of the same nephron, juxtaglomerular cells, which secrete renin, and extraglomerular mesangial cells.[21](Figure 7)

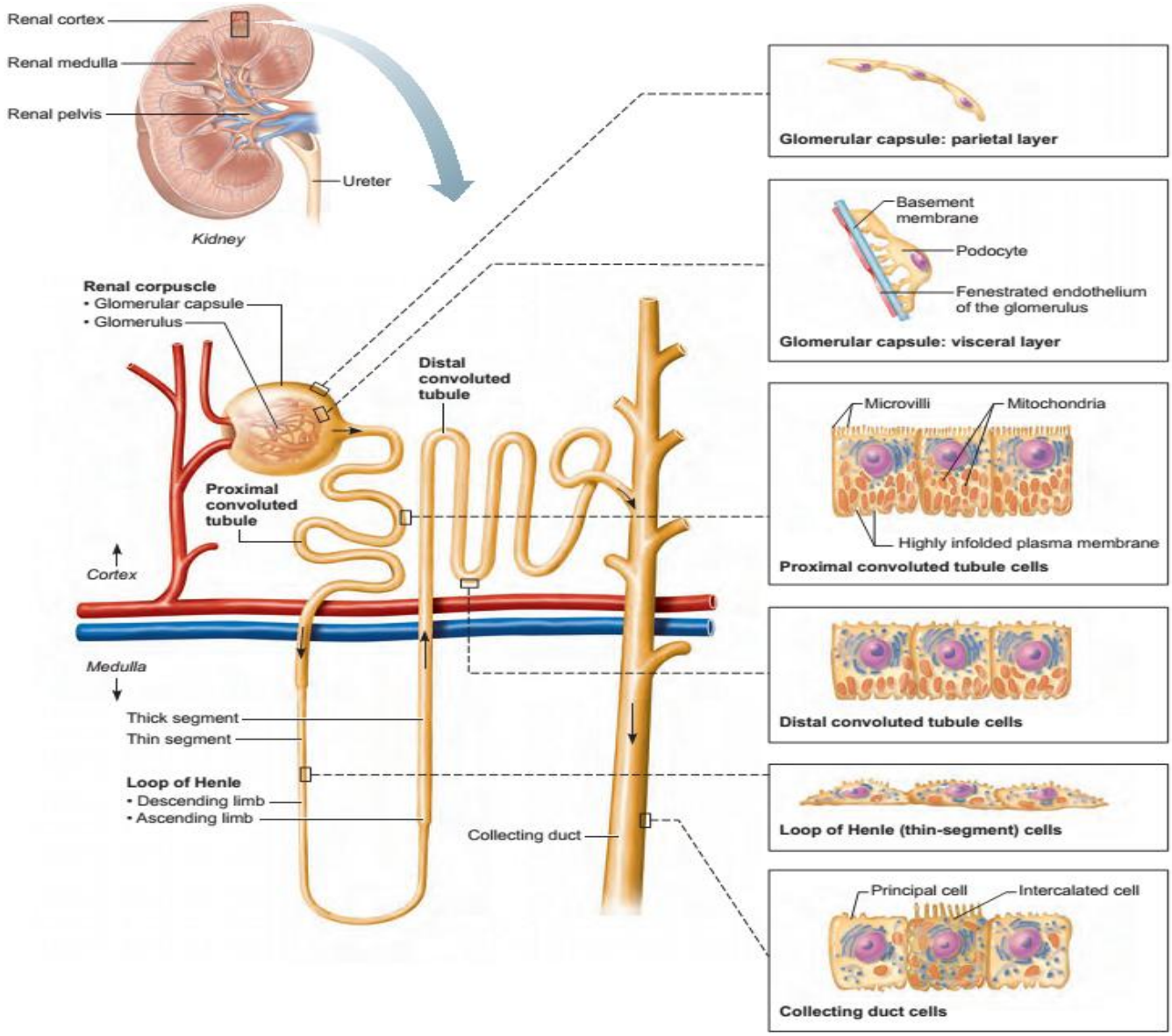


Figure 5 : Location and structure of nephrons. Schematic view of a nephron depicting[17] and the structural characteristics of epithelial cells forming its various regions.

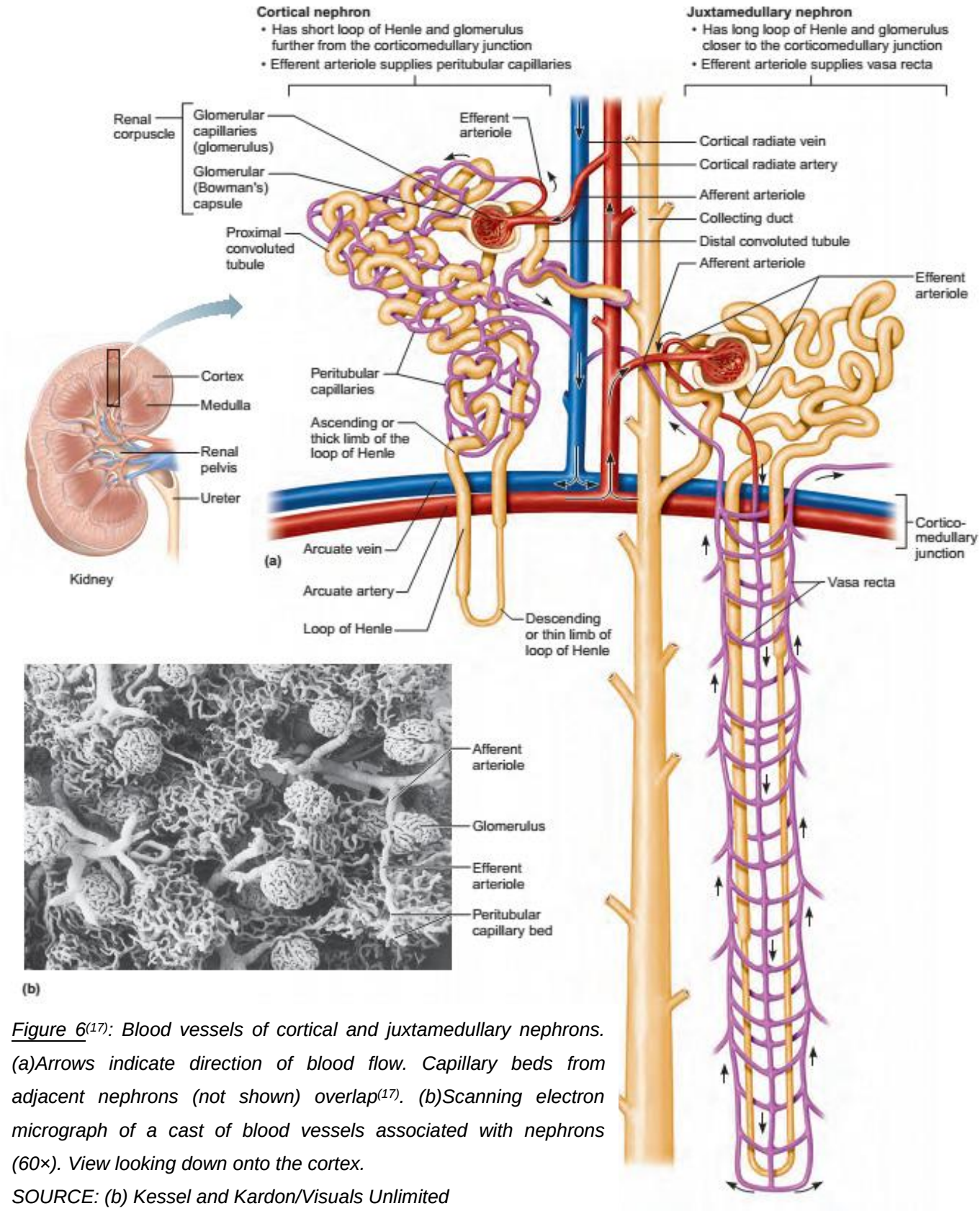


Figure 6[17]: Blood vessels of cortical and juxtamedullary nephrons. (a)Arrows indicate direction of blood flow. Capillary beds from adjacent nephrons (not shown) overlap[17]. (b)Scanning electron micrograph of a cast of blood vessels associated with nephrons (60×). View looking down onto the cortex.

SOURCE: (b) Kessel and Kardon/Visuals Unlimited

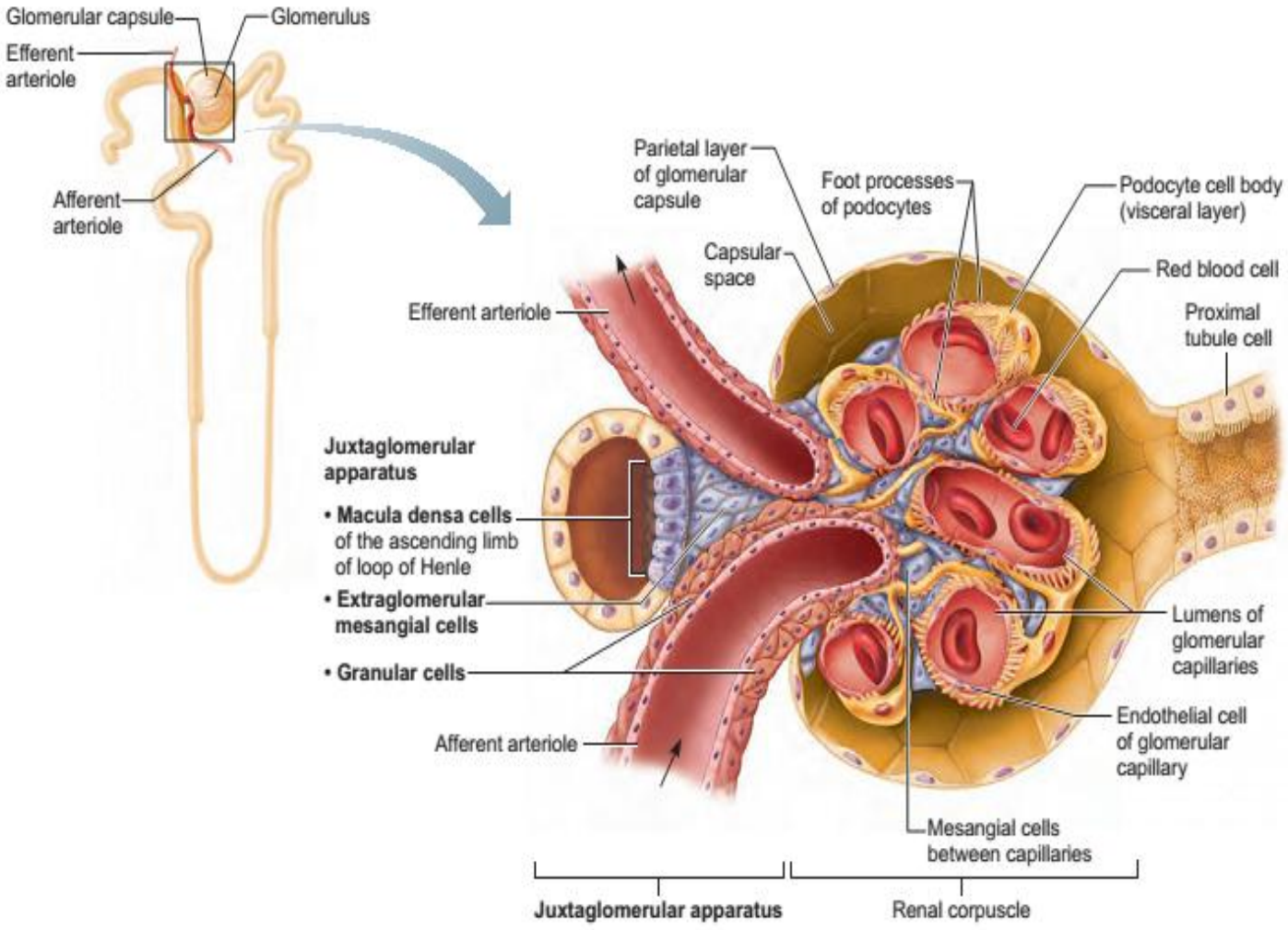


Figure 7[17]: Juxtaglomerular apparatus (JGA) of a nephron. Mesangial cells that surround the glomerular capillaries (glomerular mesangial cells) are not part of the JGA

1.3 Blood supply :

- The renal arteries branch directly from the abdominal aorta and enter the kidneys through the renal hilus.
- Inside our kidneys, the renal arteries diverge into the smaller afferent arterioles of the kidneys.
- Each afferent arteriole carries blood into the renal cortex, where it separates into a bundle of capillaries known as a glomerulus.
- From the glomerulus, the blood recollects into smaller efferent arterioles that descend into the renal medulla.
- The efferent arterioles separate into the peritubular capillaries that surround the renal tubules.
- Next, the peritubular capillaries merge to form veins that merge again to form the large renal vein.
- Finally, the renal vein exits the kidney and joins with the inferior vena cava, which carries blood back to the heart[18].(Figure 8)

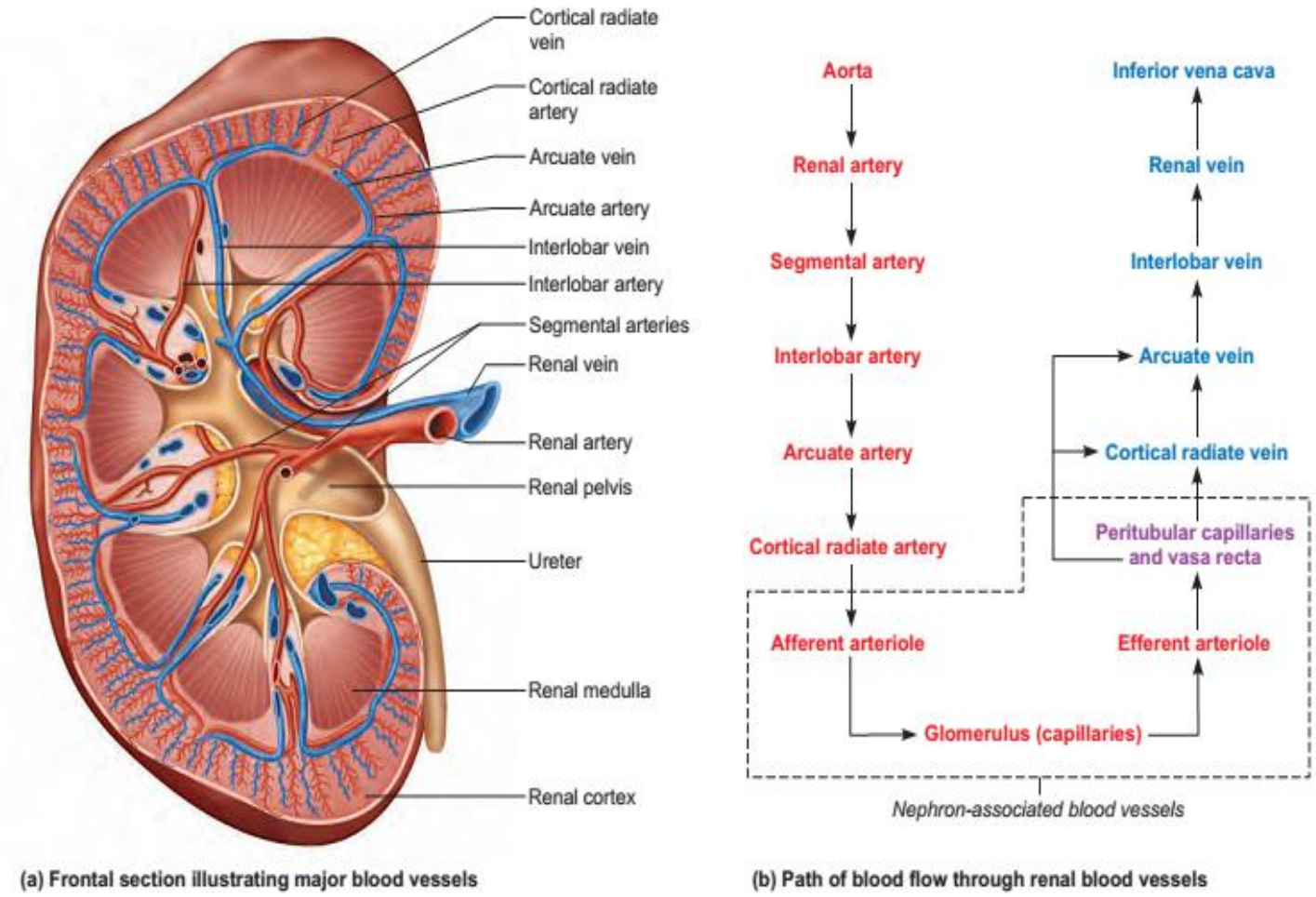


Figure 8: Blood vessels of the kidney[17].

2.Physiology of the kidney:(18)

The kidneys perform several functions, that can be classified into 2 distinguished types :

2.1 exocrine functions :

Ø Excretion of Wastes:

The primary function of the kidneys is the excretion of waste products resulting from protein metabolism and muscle contraction. Notably the toxic ammonia, uric acid and urea, that come from the dietary proteins metoblized by the liver. The same applies to creatinine, produced in the process of muscle-use of the creatine as an energy. All these harmful substances accumulate in the body over time and need to be removed from circulation to maintain homeostasis.

Ø Filtration, Reabsorption, and Secretion :

- The kidneys filter blood as it passes through the capillaries that form the glomerulus. The crossing of blood cells and plasma through the lining of the capillaries, is conditioned by the blood pressure which forces most of the blood plasma through the lining, and the size of blood cells. The filtered plasma, now known as tubular fluid, begins to flow out of the glomerular capsule and into the proximal convoluted tubule.
- At the same time, the concentrated blood that remains inside the capillaries of the glomerulus moves into the efferent arterioles and on to the peritubular capillaries surrounding the proximal convoluted tubule, where glucose, amino acids ,ions and any remaining waste products, will reabsorbed back into the blood.
- From the proximal convoluted tubule, the tubular fluid next enters the loop of Henle, where water and ions are reabsorbed. The exchanges at this level are ensured by osmotic pressure between the hypotonic filtrate and hypertonic

medullary cells, that pushes water out of the filtrate and into the cells, and then into the blood.

- Filtrate next passes through the ascending limb of the loop of Henle, where tissues are not permeable to water but are permeable to ions. The filtrate is very concentrated after passing through the descending limb, so ions easily diffuse out of the filtrate and are returned to the blood flowing through nearby capillaries.
- Tubular fluid exiting the loop of Henle next passes through the distal convoluted tubule and the collecting duct of the nephron. These tubules continue to reabsorb small amounts of water and ions that are still left in the filtrate. Meanwhile, They actively absorb excess potassium and hydrogen ions, and secrete them into the filtrate.
- When filtrate reaches the end of the collecting duct, almost all of the valuable nutrients, ions, and water have been returned to the blood supply, while waste products and a small amount of water are left to form urine. The urine exits the collecting duct and joins with urine from other collecting ducts in the renal pelvis.

Ø Water homeostasis:

The kidneys are able to control the volume of water in the body by changing the reabsorption of water by the tubules of the nephron. Under normal conditions, the tubule cells of the nephron tubules reabsorb (via osmosis) nearly all of the water that is filtered into urine by the glomerulus.

Ø Acid/base homeostasis:

The kidneys regulate the pH level of the blood by controlling the excretion of hydrogen ions (H+) and bicarbonate ions (HCO3-). Both ions are filtered out of the blood in the glomerulus of the kidney, but the tubule cells lining the nephron

selectively reabsorb bicarbonate ions while leaving hydrogen ions as a waste product in urine. The tubule cells may also actively secrete additional hydrogen ions into the urine when the blood becomes extremely acidic.

Ø Electrolyte homeostasis :

The kidneys maintain the homeostasis of important electrolytes by controlling their excretion into urine :

- *Sodium(Na+)* :Over 99% of the sodium ions passing through the kidneys are reabsorbed into the blood from tubular filtrate, especially in the proximal convoluted tubule and ascending loop of Henle.
- *Potassium (K+)* :Unlike sodium, only about 60 to 80% of the potassium ions passing through the kidneys are reabsorbed. Most of the reabsorption of potassium occurs in the proximal convoluted tubule and ascending loop of Henle.
- *Chloride (Cl-)* :The proximal convoluted tubule and ascending loop of Henle reabsorb about 90% of the chloride ions filtered by the kidneys.
- *Calcium (Ca2+)* :The proximal convoluted tubule and the ascending loop of Henle reabsorb most of the calcium in tubular filtrate into the blood. Parathyroid hormone increases the reabsorption of calcium in the kidneys when blood calcium levels become too low.
- *Magnesium (Mg2+)* :The proximal convoluted tubule and loop of Henle reabsorb most of the magnesium that passes through the kidney.

Ø Blood pressure homeostasis:

The kidneys help to control blood pressure in the body by regulating the excretion of sodium ions and water and by producing the enzyme renin (endocrine function). The kidneys are able to control blood pressure by either reabsorbing

water to maintain blood pressure or by allowing more water than usual to be excreted into urine and thus reduce blood volume and pressure.

2.2 Endocrine functions:

The kidneys maintain a small but important endocrine function by producing the hormones calcitriol and erythropoietin:

- *Renin* , a hormone that it exclusively synthesized by the kidney, it allows the produce of angiotensin II from angiotensinogen and aldosterone, hormones that are involved in the regulation of the pressure blood.
- *Erythropoietin (EPO),* a hormone produced by cells of the peritubular capillaries in response to hypoxia. EPO stimulates the cells of red bone marrow to increase their output of red blood cells, but Once oxygen levels return to normal, the cells of the peritubular capillaries stop producing EPO.
- *Calcitriol* , the active form of vitamin D in the body. Tubule cells of the proximal convoluted tubule produce calcitriol from inactive vitamin D molecules. At that point, calcitriol travels from the kidneys through the bloodstream to the intestines, where it increases the absorption of calcium from food in the intestinal lumen.

Chronic kidney disease:

1. Definition & Staging:

- In the United States, the national kidney foundation (NKF-KDOQI) guidelines, defines CKD as<<either a state of kidney damage or a GFR <60mL/min/1.73m^2 for more than 3 months, and kidney damage is defined as pathologic abnormalities or markers of damage, including abnormalities in blood or urine tests or imaging studies[22][23]>> .Tables 1 summaries the classification and treatment goals for the 5 stages of CKD according to KDOQI guidelines. The higher CKD stage, the more severe the renal insufficiency.
- While, in France, the National Agency for Accreditation and Health care evaluation (ANAES), specifies 2 separated entities[24][25] :
  - *The* chronic *kidney failure*, that indicates a permanent and progressive reduction of the glomerular filtration rate (GFR)below 60mL/min/1.73m^2 for over a period of 3 months, with or without kidney damage markers. In table 2 , the different stages of CKD and levels of kidney function are described.
  - *The chronic kidney disease*, in which GFR is greater than 60ml /min/1.73^{m2} while the markers of damage are present for more than 3 months..
- In both definitions, CKD has to be distinguished from acute renal failure (ARF), where GFR decreases transitorily and is reversible at most of the time.
- In the course of CKD, the permanent, and slow deterioration of the kidneys function, is actually related to an irreversible loss of significant number of functional nephrons. In fact, chronic kidney failure signify that at least the half

kidney's nephrons are no longer functional, and by the end-stage-renal-disease (ESRD),the kidney have only10% of functional nephrons(26).

- CKD still a long silent and crippling disease, characterized by a progressive course of worsening renal function eventually leading to end-stage renal disease (ESRD), which requires renal replacement therapy (RRT) by means of dialysis or kidney transplantation.

Table 1 : CKD stages according to NKD-KDOQI guidelines.(22)(23)

Stage	GFR (mL/min/1.73m^2)	Description	Treatment goal
1	≥90	Renal damage with normal or ↑ GFR	Diagnosis, treatment of co-morbid conditions, slowing progression, cardiovascular disease risk reduction
2	60–89	Renal damage with mild ↓ GFR	Estimate progression
3	30–59	Moderate ↓ GFR	Evaluate and treat complications
4	15–29	Severe ↓ GFR	Preparation for renal replacement therapy
5	<15 (+ dialysis)	Renal failure	Renal replacement therapy (dialysis) if uraemia present

Table 2 : CKD stages according to the national agency of accreditation and health care evaluation, guidelines.(25)

Stage	GFR	description
1	DFG>60 ml/min	Chronic kidney disease with a GFR>60ml/min
2	30≤DFG<59 ml/min	Moderate CKD
3	15≤DFG<29 ml/min	Severe CKD
4	DFG<15 ml/min	ESRD

2. Epidemiology :

2.1 Descriptive epidemiology:

a) World global facts :

- It is estimated that 10% of the population worldwide is affected by chronic kidney disease, and In people aged 65 through 74 worldwide, one in five men, and one in four women, have CKD. (27)
- According the 2010 Global Burden of Disease study, chronic kidney disease was ranked 18th in the list of causes of total number of deaths worldwide, while it was ranked as the 27th in 1990 (28).
- Chronic kidney disease is a worldwide health crisis. For example, in the year 2005, there were approximately 35 million deaths worldwide, attributed to chronic disease, according to the World Health Organization. (29)
- Over 2 million people worldwide currently receive treatment with dialysis or a kidney transplant to stay alive, yet this number may only represent 10% of people who actually need treatment to live.(30)
- Of the 2 million people who receive treatment for kidney failure, the majority are treated in only five countries – the United States, Japan, Germany, Brazil, and Italy. These five countries represent only 12% of the world population. Only 20% are treated in about 100 developing countries that make up over 50% of the world population.(30)
- In middle-income countries, over 1 million people die annually from untreated kidney failure(30).

b) Moroccan facts:

- According to 2012 KDIGO guidelines, prevalence of CKD in Morocco was estimated to be 5.1%.The distribution over KDIGO stages was the following :

CKD1: 17.8%; CKD2: 17.2%; CKD3: 52.5% (3A: 40.2%; 3B: 12.3%); CKD4: 4.4%; CKD5: 7.2%.[31]

- The same prevalence (5.1%) was brought out in MAREMAR cross-sectional study, that was based on stratified random sample of the adult population of two Moroccan towns (El jadida and Khemisset). [32]
- Only about 250 patients have a kidney transplant in Morocco [33].
- According to Pr. Driss Zaid, previous president of the Moroccan Society of Nephrology, the estimated cases of ESRD in Morocco are 100 to 120 people on 1 million. And about 3,000 reported cases of ESRD are still waiting for dialysis each year.[33]
- Unlike the worldwide facts tendency, CKD in morocco is 2 to 3 times more common in men than women. [33]

c) Other countries:

- The prevalence of CKD in general population in the US and in Europe is about 10% of adults [34][35]. The prevalence of early stages of the CKD disease (CKD1-4, 3.3%, 3.0%, 4.3% and 0.2% respectively) is 100 times higher than the prevalence of end-stage kidney disease (G5 0.1%) [36].
- In the US, treatment of chronic kidney disease is likely to exceed $48 billion per year. Treatment for kidney failure consumes 6.7% of the total Medicare budget to care for less than 1% of the covered population [27].
- In China, the economy will lose US$558 billion over the next decade due to effects on death and disability attributable to heart disease and kidney disease.[27]
- In England, according to a recent report published by NHS Kidney Care, chronic kidney disease costs more than breast, lung, colon and skin cancer combined.[27]

ü In Australia, treatment for all current and new cases of kidney failure through 2020 will cost an estimated $12 billion. (27)

2.2 Analytical epidemiology:

Ø CKD risk factors :(37)(38)(39)

- CKD risk factors are incriminated not only in the development of the disease its self, but also in the progression of renal impairment and the development of complications, reason why they are the first target of CKD treatment. Their prevalence and severity increase as long as kidney function declines. CKD risk factors are considered as being also the causes of chronic renal failure, they can be divided into traditional risk factors and non-traditional risk factors.
- Traditional risk factors (tables 3a-3b), are risk factors that have been defined and validated in prospective studies in the general population, the most incriminated ones are:
  - § Diabetes : Considered as the most common risk factor of CKD, in fact, the high blood sugar in patients with diabetes may damage the blood vessels in kidney's nephrons or glomerulus, then leads to renal insufficiency.
  - § High blood pressure (HBP) : there is a strong relationship between HBP and CKD development, since it is responsible of kidney failure at 25% of patients with HBP, In fact, hypertension may damages, the small blood vessels of renal tubule, leading to a quick deterioration of nephrons and hence to failure. Medical treatment, a proper diet, weight control and physical exercise, still the best means for HBP prevention, in in order to preserve kidneys in healthy status

- Non-traditional risk factors (Table 4), are those that occur as a consequence of impaired renal function (e.g anemia, cardiovascular diseases..), or others, such as infection, inflammation, genetic factors...

Table 3 a : Traditional risk factors of CKD and cardiovascular disease in the course of CKD.

Risk factors	Chronic kidney disease		Cardiovascular disease (development of)	Mortality
	development of	progression		
Increased in age	+	unconfirmed	+	+
Ethnicity (Australian aboriginal/ African American)	+	unconfirmed	+	+
Exposure to nephrotoxic drugs / chemicals	+	+	Not established	Not established
Cardiovascular disease/ left ventricular hypertrophy	+	unconfirmed	n/a	+
Diabetes mellitus (glycaemic control)	+	+	+	+
Dyslipidaemia – ↑ LDL cholesterol	unconfirmed	unconfirmed	+	+
Dyslipidaemia – ↓ HDL cholesterol	unconfirmed	unconfirmed	+	+
Dyslipidaemia – ↑ triglycerides	unconfirmed	unconfirmed	+	+
Family history of heart disease	Not established	Not established	+	unconfirmed
Family history of kidney disease	+	+	Not established	unconfirmed
Gender (male)	+	+	+	unconfirmed
History of acute kidney injury (AKI)	+	+	Not established	Not established
Hypertension	+	+	+	+
Kidney/urinary tract stones	unconfirmed	unconfirmed	Not established	Not established
Menopause	Not established	Not established	+	Not established

table 3 b (continued) :Traditional risk factors of CKD and cardiovascular disease in the course of CKD.(Following)

Metabolic syndrome (MetS)	+	unconfirmed	+	unconfirmed
Nephron mass (low), e.g., low birth weight, nephrectomy	+	+	Not established	Not established
Obesity	+	+	+	+
Physical inactivity	+	unconfirmed	+	Not established
Proteinuria/Albuminuria	+	+	+	+
Psychosocial stress/ disadvantages	+	unconfirmed	+	Not established
Smoking	+	+	+	+
Urinary tract infection	+	+	Not established	Not established
Urinary tract obstruction	+	+	Not established	Not established

Table 4 : Non-traditional risk factors of CKD and cardiovascular disease in the course of CKD.

Risk factors	CKD progression	Cardiovascular disease Risk factors altered by CKD	Mortality
↓ s-albumin	unconfirmed	unconfirmed	+
Anaemia	+	+	+
Bone mineral disorders/ ↑ calcium x phosphate products	+	+	+
Cardiovascular disease	+	n/a	+
CKD, ↓ GFR/ stage CKD	n/a	+	+
Dyslipidaemia – ↑ LDL cholesterol, ↓ HDL cholesterol and ↑ triglycerides	unconfirmed	+	+
Electrolyte imbalance	+	+	unconfirmed
Family history of CKD	+	Not established	unconfirmed
↑ Homocysteine	unconfirmed	unconfirmed	unconfirmed
Inflammation/ infection	+	+	unconfirmed
Malnutrition/ protein and energy wasting (PEW)	unconfirmed	+	unconfirmed
Oxidative stress	unconfirmed	+	unconfirmed
Proteinuria/ albuminuria	+	+	unconfirmed
↑ Renin-angiotensin system activity	+	+	unconfirmed
Thrombogenic factors	unconfirmed	+	unconfirmed
Uraemic toxicity	+	Not established	unconfirmed

3. Assessment of Kidney Function :

Accurate estimation of kidney function is central to the detection, evaluation, and treatment of chronic kidney disease (CKD). Glomerular filtration rate (GFR) is widely accepted as the best overall measure of kidney function and as such it forms the basis of definition and classification system for CKD. GFR cannot be measured directly in clinical practice and multiple methods are used to estimate GFR, all of which have their strengths and limitations. The current recommended method is to use estimating equations based on serum creatinine and which incorporate demographic and clinical variables[40].

3.1 markers of renal function tests:

The markers of renal function test assess the normal functioning of kidneys. These markers may be radioactive and non radioactive. They indicate the glomerular filtration rate, concentrating and diluting capacity of kidneys (tubular function). If there is an increase or decrease in the valves of these markers it indicates dysfunction of kidney[41].

Creatinine, urea, uric acid and electrolytes are makers for routine analysis whereas several studies have confirmed and consolidated the usefulness of markers such as cystatin C and β-Trace Protein[41].

1. Creatinine:

A breakdown product of creatine phosphate in muscle[42]. Commonly used as measure of kidney function. The normal creatinine clearence test valve is 110-150ml/min in male and in female it is 100-130ml/min[43]. The National Kidney Disease Education Program recommends calculating glomerular filtration rate from serum creatinine concentration[44]. The diagnosis of renal failure is usually suspected

when serum creatinine is greater than the upper limit of the “normal” interval. However, an increased tubular secretion of creatinine in some patients could give false negative value, since creatinine may be influenced by muscles function, muscles composition, activity, diet[45][46], and many other illness such as, muscular dystrophy paralysis, anemia, leukemia and hyperthyroidism.[47]

2. Urea:

Major nitrogenous end product of protein and amino acid catabolism.[43] It is useful in differential diagnosis of acute renal failure and pre renal condition where blood urea nitrogen–creatinine ratio is increased[48]. Urea clearance is a poor indicator of glomerular filtration rate as its overproduction rate depends on several non renal factors, including diet and urea cycle enzymes.[49]

3. Cystatin C:

The protease inhibitor Cystatin C is a non-glycosylated low molecular weight protein, that has been, recently, proposed asa marker of glomerular filtration rate, that is superior to serum creatinine[50], it was also found to be an effective marker for GFR in patients with cirrhosis following liver transplantation[51][52]. Cystatin C has been found more useful for detecting early renal impairment in both type 1 and type 2 diabetic patients.[53] . Moreover Cystatin C was also found to be associated with mild kidney dysfunction with increased risk for cardiovascular events, peripheral arterial disease and heart failure.[54]

4. β -Trace Protein (BTP) :

β-Trace Protein is a low-molecular weight glycoprotein, which has been reported to be a better indicator of reduced glomerular filtration rate than serum creatinine. [55][56] Serum β -Trace Protein has been found to be elevated in patients with renal diseases.[57] However, when compared, Cystatin C is still a better indicator than Serum β-Trace Protein[58].

5. Inulin :

Fructose polymer inulin (MW 5kDa) satisfies the criteria as an ideal marker of glomerular filtration rate. Rapid measurement of glomerular filtration rate by an inulin single-bolus technique would be practically useful. (59)

3.2Glomerular filtration rate (GFR):

- The GFR is the volume of fluid filtered from the renal glomerular capillaries into the Bowman's capsule per unit time (60), namely, it is the product of the filtration rate in single nephrons and the number of nephrons in both kidneys.
- Thus, Reductions in GFR can be caused by either a decline in nephron number (as in CKD) or by a decline in single nephron GFR, which is due to physiologic or pharmacologic factors causing hemodynamic alterations in glomerular filtration .
- An increase in single nephron GFR caused by increased glomerular capillary pressure or glomerular hypertrophy can compensate for a decrease in nephron number, and the level of GFR may not reflect the loss of nephrons. That is to say, that substantial kidney damage may be present before GFR declines.(40)
- In adults, the normal GFR number is more than 90. GFR declines with age, even in people without kidney disease. (See the table 3 below for average estimated GFR based on age).
- Different formulas have been proposed for GFR estimation, the most useful ones in practice, are the following :

- *Cockcroft and Gault Formula :* (61)(62)

$$eC_{Cr} = \frac{(140 - \text{Age}) \times \text{Mass (in kilograms)} \times Constant}{8.8 \times \text{Serum Creatinine (in mg/dL)}}$$

- Constant = 1.04 if Female / 1.23 if Male

- *Modification of diet in renal disease (MDRD)* (63)

$$\text{eGFR} = 186 \times \text{Serum Creatinine}^{-1.154} \times \text{Age}^{-0.203} \times [1.210\ if\ Black] \times [0.742\ if\ Female]$$

- *Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) Formula*(64)

$$\text{eGFR} = 141 \times \min(\text{SCr}/k,1)^{a} \times \max(\text{SCr}/k,1)^{-1.209} \times 0.993^{Age} \times [1.018\ if\ Female] \times [1.159\ if\ Black]$$

- Where SCr is serum creatinine (mg/dL), k is 0.7 for females and 0.9 for males.
- a is -0.329 for females and -0.411 for males.
- min indicates the minimum of SCr/k or 1
- max indicates the maximum of SCr/k or 1

- *Schwartz formula for children:* (65)(66)(67)

$$\text{eGFR} = \frac{k \times Height}{Serum\ Creatinine}$$

- The constant k vary according to the child's mass :

  K = 26 if Mass < 2 kg

  k = 29 if 2kg < Mass < 3kg

  K = 35 if 5kg < Mass < 12kg

  K = 49 if girl with a body mass > 12kg or boy 12kg < Mass < 42kg

  K = 53 if boy with a body mass > 42kg

Table 5 : Estimated GFR values based on age, according to national kidney foundation [68].

Age (years)	Average estimated GFR
20-29	116
30-39	107
40-49	99
50-59	93
60-69	85
70+	75

4. Causes of CKD :

- Many causes of CKD exist, but the most prevalent causes include hypertension, diabetes, glomerulonephritis and urinary tract obstructions[69].
- Historically, kidney disease has been classified according to the part of the kidney anatomy involved[70] :
  - Vascular disease includes large vessel disease such as bilateral renal artery stenosis and small vessel disease such as ischemic nephropathy, hemolytic-uremic syndrome, and vasculitis.
  - Glomerular disease comprises a diverse group and is classified into:
    - Primary glomerular disease such as focal segmental glomerulosclerosis and IgA nephropathy (or nephritis)
    - Secondary glomerular disease such as diabetic nephropathy and lupus nephritis
  - Congenital disease such as polycystic kidney disease.

- Tubulointerstitial disease includes drug- and toxin-induced chronic tubulointerstitial nephritis, and reflux nephropathy.
- Obstructive nephropathy is exemplified by bilateral kidney stones and diseases of the prostate.
- On rare cases, pinworms infecting the kidney can also cause nephropathy

5. pathogenisis of CKD :

ü Once half of the total nephrons are lost, CKD progresses similarly regardless of etiology. Indeed, initial hyperfiltration activates Renin angiotensin aldosterona system (RAAS) and causes proteinuria. Angiotensine II and protein uptake at the tubules causes inflammation and fibrosis of the glomerulus and tubules. Progressive decline in the GFR and systemic complications occurs[71 - 76]. (Figure 9)

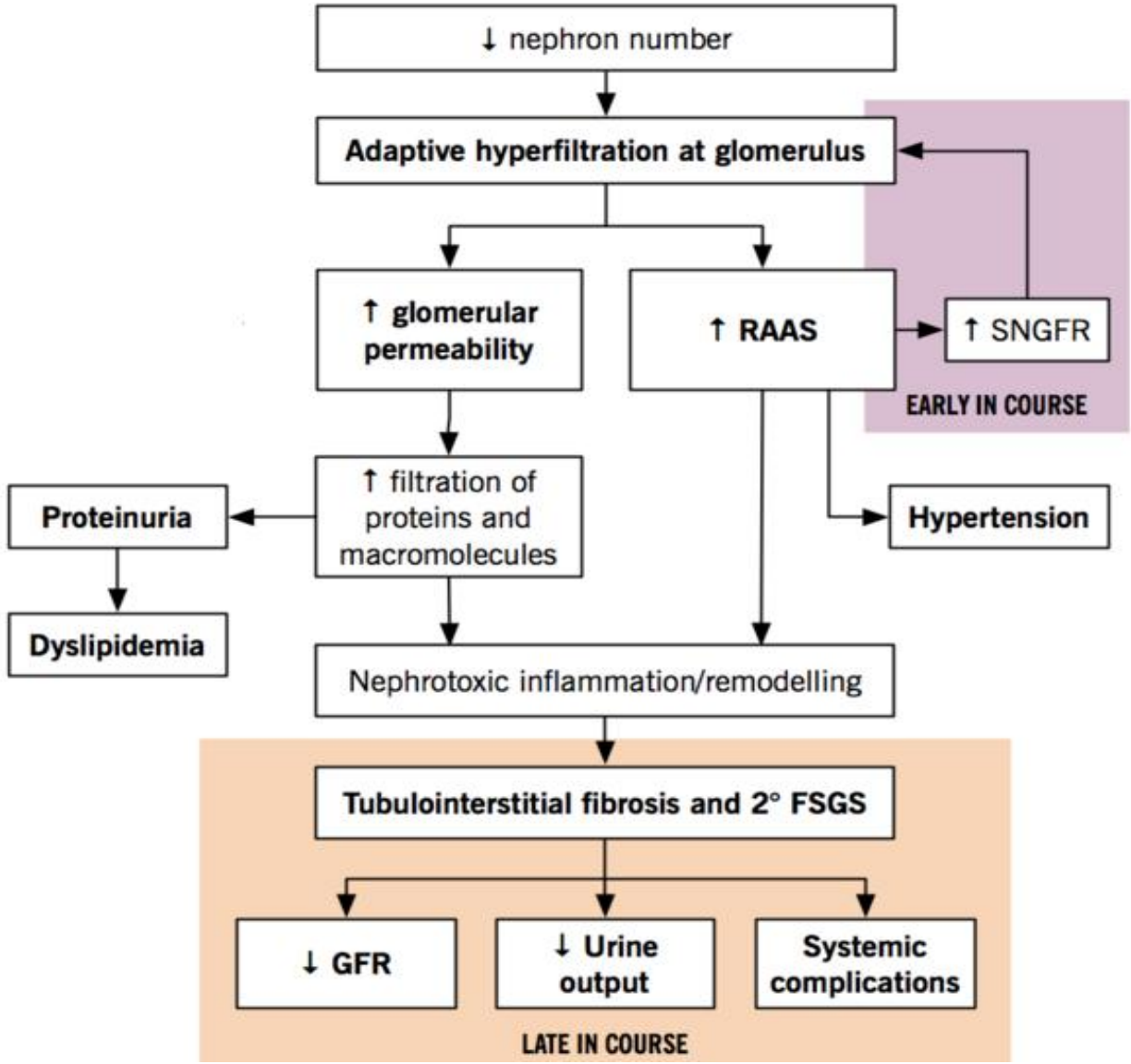


Figure 9 :Pathogenisis of chronic kidney disease.

FSGS: Focal segmental glomerulosclerosis

SNGFR :Single nephron GFR

5.1 Hypertension:[71]

- Glomerular and vascular changes:
  - Elevated systemic blood pressures cause a hypertrophic response leading to intimal thickening of the large and the small vasculature.
  - The mechanisms are compensatory at first, but later lead to glomerular damage
    - Global sclerosis – ischemic injury to the nephrons causes death
    - Focal segmental sclerosis – glomerular enlargement for compensation of the loss of nephrons in other areas of the kidney.
- Interstitialnephritis:
  - The vascular and glomerular disease lead to tubular atrophy and an intense chronic interstitial nephritis
    - The intense chronic interstitial nephritis is thought be secondary to immunologic processes against ischemia-mediated antigen changes on the tubular epithelial cell surface.
- Chronically these changes lead to tubular and glomerular loss causing nephrons loss.
  - With the death of some nephrons, less are available to maintain the GFR.
  - Gradual decline in the GFR is noticed as the nephrons continue to die.

5.2 Diabetes – diabetic nephropathy:[72][73]

- Chronic hyperglycemia is thought to be the primary cause of diabetic nephropathy.
  - Unlike other tissues of the body, transmembrane glucose transporters (GLUT) receptors do not facilitate intracellular glucose transport in the kidneys.

- This effect is mediated via a number of mechanisms including (i) glomerular hyperfiltration, (ii) direct effects of hyperglycemia, and (iii) advanced glycosylation end products (AGE), and (iv) cytokine secretion.

- *Glomerular hyperfiltration*:
  - Glomerular hyperfiltration is mediated mainly via dilatation the afferent arteriole leading to a rise in the GFR and the renal blood flow.
  - This dilatation of the afferent arteriole is mediated by a number of mechanisms in diabetic nephropathy:
    - Hyperglycemia and high insulin-like growth factor-1 (IGF-1) concentrations (observed in diabetic patients) – both are hypothesized to cause a rise in the GFR increasing renal flow
    - Hyperfiltration of glucose leads to augmented sodium-glucose transport in the proximal convoluted tubule causing enhanced sodium transport
      - Cause expansion of blood volume which leads to a rise in GFR
      - The rise in proximal reabsorption also leads to a reduced distal fluid delivery which activates the tubuloglomerular feedback with the renin-angiotensin system which works to raise the GFR as well.

- Hyperglycemia and AGE:
  - Hyperglycemia and AGE directly induce mesangial matrix production, cellular expansion and apoptosis.
  - The two have also been shown to increase basement membrane permeability to albumin.

- Cytokines:
  - Elevations in vascular endothelial growth factor (VEGF), transforming growth factor beta (TFG-β), and profibrotic proteins increase damage to the nephrons at different levels; specific mechanisms are unclear. (figure 10)

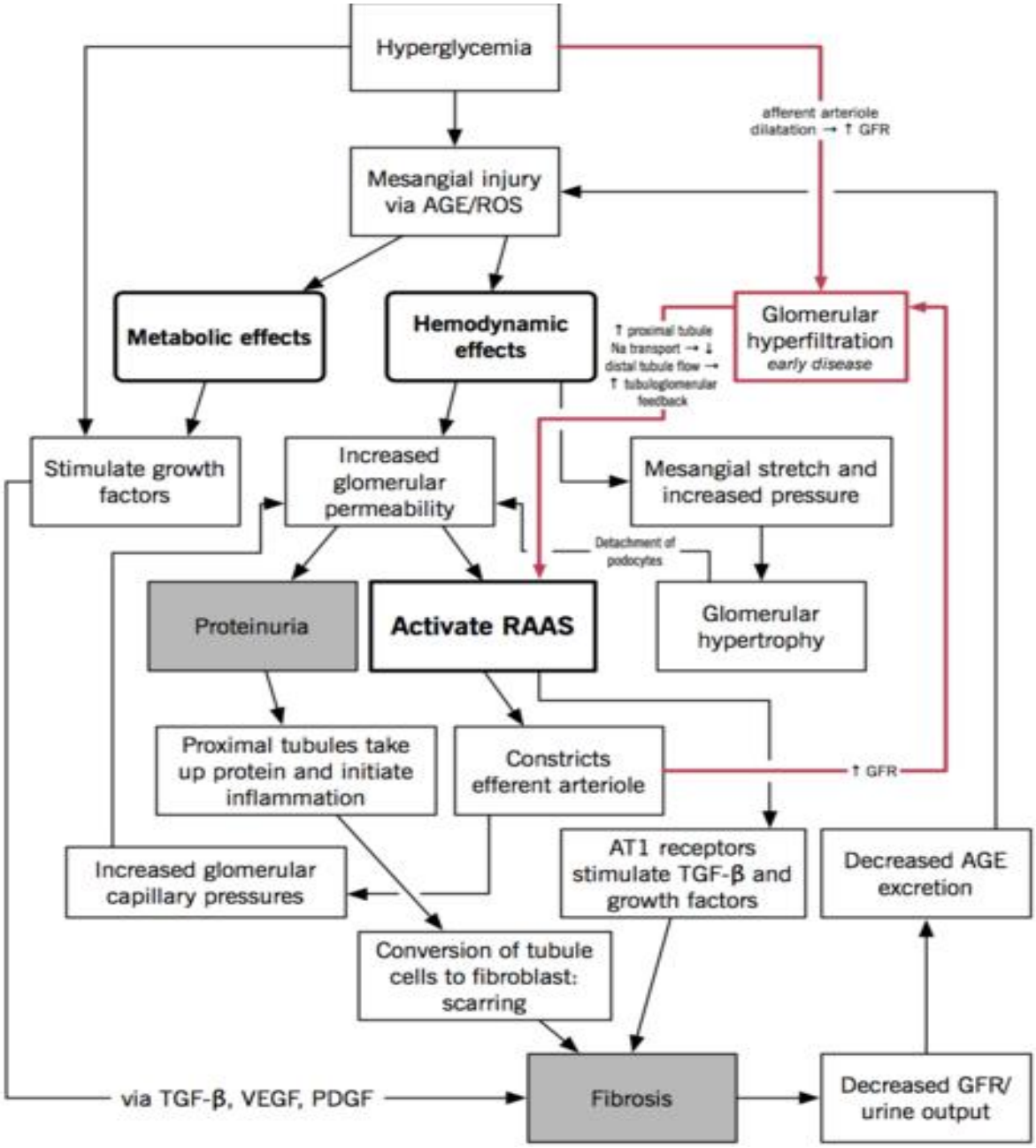


Figure 10 : Pathogenesis of diabetic nephropathy

5.3Glomerulonephritis:[74][75][76]

- Glomerular injury takes place via inflammatory as well as non-inflammatory mechanisms in different types of glomerulonephritis.
- Non-inflammatory in jury:
  - Common examples of this include the podocytopathies; minimal change nephrotic syndrome/focal segmental glomerulo sclerosis (MCNS/FSGS) and membranous nephropathy(MN)
    - A dramatic rise in the glomerular permeability without any evidence of inflammation on light microscopy.
    - Several cytokines such as IL-13 and members of the complement system C3, C5b-9 lead to glomerular basement membrane thickening, as well as podocyte damage, apoptosis, detachment and excretion in the urine. This induces glomerularsclerosis.
- Inflammatory injury:
  - Common examples include post streptococcal glomerulonephritis (GN) membrano proliferative GN, Henoch-Schönlein purpura (HSP), systemic lupus erythematosus (SLE), some forms of rapidly progressive glomerulonephritis (RPGN), IgA nephropathy, hemolytic uremic syndrome (HUS) and various vasculitides.
  - This inflammatory injury has been found to be mediated by a number of mechanisms:
    - Some members of the complement system such as C5a have been implicated in inflammatory injury via inducing antibody deposition and activation and recruitment of polymorphonuclear cells (PMNs); neutrophil, macrophage/monocyte, platelets and T-cells.

- These cells produce oxidants and proteases that cause fibrin deposition, capillary wall damage and produce proteinuria.
- Unlike podocyte targeting in non-inflammatory injury, disorders in which glomerular endothelial and mesangial cells are principally involved exhibit a more dramatic response to immune injury. This response is usually characterized by cell proliferation and phenotype change, as well as readily visible structural changes in the renal biopsy.

6. CKD diagnosis :

6.1 Clinical features:

Clinically, chronic kidney disease remains asymptomatic for a long time before getting discovered. In fact, it may be discovered during a routine health checkup, when a complication is developed, during the follow-up of a nephropathy or a systemic disease such as diabetes, or when these CKD related symptoms are present, they are actually multiple and variable from one individual to another :

- General signs: asthenia, anorexia..
- Renal signs: eodema, micturition disorder
- Central or peripheral neurologic symptoms
- Gastrointestinal manifestations: nausea, vomiting
- Cardiovascular events: hypertension, pericarditis
- Hematological manifestations: anemia, bleeding disorders
- Endocrine and systemic manifestations: hormonal disorders,

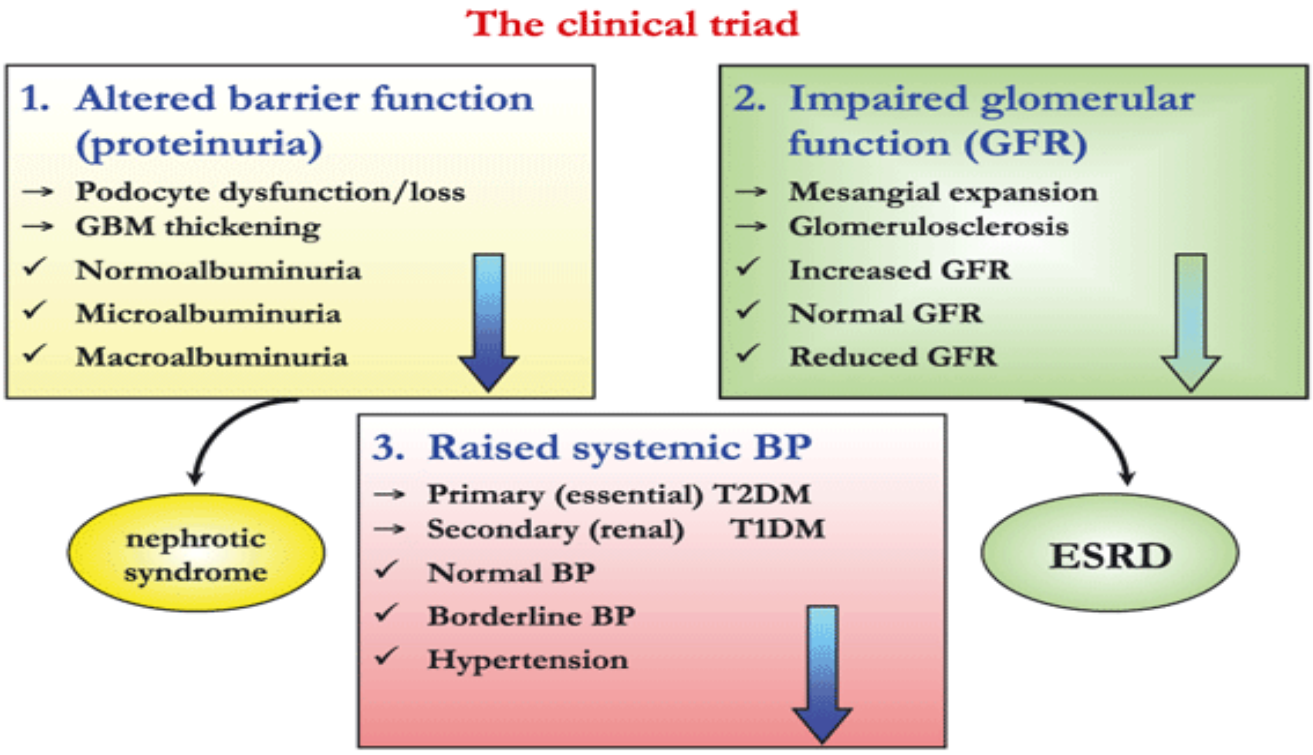


Figure 11: Clinical features of CKD

6.2 Work-up:

Ø To confirm diagnosis:[77]

In order to confirm the CKD diagnosis, we need to :

- ü Order a first Serum creatinine determination, which should be elevated.
- ü Estimate the creatinine clearance and calculating the GFR , often using the Cockcroft-Gault formula. A lowered value of GFR less than 60mL/min should be found out.
- ü Look for creatinine variation factors, since it may be influenced in unpredictable ways by assay techniques, endogenous and exogenous substances, renal tubular handling of creatinine, and other factors (age, sex, body weight, muscle mass, diet, drugs).

- A second serum creatinine determination is usually recommended (preferably in the same Lab), and hence a second determination of GFR.

Ø To confirm chronicity:[78]

The chronicity of renal failure is defined by a decreased kidney function for over 3 months; where many blood disorders and morphologic abnormalities are already developed:

- Normochromic normocytic anemia with a hemoglobin < 12g/dL .
- Hypocalcemia, due to active vitamin D deficiency related to absence of renal hydroxylation in alpha-1 position.
- Decrease in kidney size (largeaxis <10 cm on ultrasound or <3 vertebrae on an abdominal X-ray, a CT scan or MRI. However, this criterion is not always found out, since kidney size can be maintained or even increased in some medical conditions : amyloidosis, diabetes, polycystic kidney disease...

Ø To seek for an etiology:

a. Diabetic nephropathy : [79]

- Diabetic nephropathy is a serious complication of diabetes mellitus.

It usually occurs after about 20 years of type 1 diabetes development, but only 20-30% of patients have nephropathy. This suggests that exposure to chronic hyperglycemia is a necessary but not sufficient condition to develop this complication, and so there's likely a predisposing genetic factors (familial history of hypertension..).

- The diagnosis of incipient nephropathy, is based on finding out an abnormal albumin excretion, that is >30 but<300 mg / 24h in two determination, on 3 samples. The clinical stage of nephropathy is

defined by a proteinuria> 300 mg / 24h, which is associated with a decreased glomerular filtration rate and high blood pressure.

b). Vascular nephropathies :

- ü Over the past 3 decades, the prevalence of vascular nephropathies, as an underlying cause of CKD, has increased. Two major vascular damage are distinguished: (80)

- o *Atherosclerotic renal artery stenosis:* they are frequent, often bilateral and had a progressive course.(81)They are also considered as a particular risk group in atherosclerotic patients. On the other hand, It is brought out that the atherosclerotic plaques sitting on the renal arteries are almost always developed in patients with severe vascular disease. Unfortunately, the damage of renal arteries, involves, in a significant number of cases, deterioration of visceral arteries as well, whose obstruction could be fatal.(82)
- o *Nephroangiosclerosis:*(83) is a nephropathy resulting from prolonged high blood pressure, that was usually non treated or uncontrolled. It actually results in a slowly progressive renal failure, which can progress to rend-stage renal disease. The diagnosis of nephroangiosclerosis is often taken for granted when histological evidences are not available. The concept of this simple filiation is increasingly challenged. Two quite different affection are listed under this term:

  -The Most common, is the “benign” nphroangiosclerosis, a tardy consequence of prolonged hypertension, which is usually not or poorly treated. It gradually evolves into CKD.

  - The “malignant” nephroangiosclerosis, completely different, It occurs acutely, in the course of a "malignant" or “accelerated” hypertension.

Kidney failure progresses very quickly in a few weeks to the end-stage, and it is usually irreversible.

c). Glomerular nephritis:[84]

- Glomerulonephritis are the best known kidney disease. Indeed, among the treated kidney disease leading to ESRD, the proportion of glomerulonephritis has declined steadily during the last 30 years, but this reduction is relative seeing that the frequency of renal vascular and diabetes has increased markedly. They affect mainly young adults.
- The most common chronic glomerulonephritis leading to ESDR is the igA nephropathy. Moreover the rapidly progressive glomerulonephritis, also called glomerulonephritis with extra capillary proliferation which is characterized by its fast evolution and which may be primary or secondary.

d). Hereditary kidney disease:

- Approximately 5-8% of patients with ESRD have a form of hereditary kidney disease.[85]
- Polycystic kidney disease (PKD) is the most common hereditary disease.
- The Alport syndrome is characterized by the association of a gradual haematuric nephropathy with ultra structural abnormalities and immunohistochemical of glomerular basement membranes, also a gradual change of sensorineural hearing loss and sometimes ocular abnormalities.[86]

7. Progression of CKD :

7.1 Risk factors of CKD progression:

Chronic Kidney Disease (CKD) may arise due to a multitude of different insults to renal function. However despite the wide range of pathological processes that may induce renal injury, substantial loss of nephrons provokes a common syndrome characterized clinically by systemic hypertension, proteinuria and a progressive decline in glomerular filtration rate (GFR) and patho-physiologically by progressive interstitial fibrosis, peritubular capillary loss with hypoxia and destruction of functioning nephrons because of tubular atrophy[87]. Extensive studies suggest that the rate of loss of GFR, that is the rate of progression of CKD, may be largely due to common secondary factors, often unrelated to the initial disease [88].

Some of these factors such as age and race are not open to intervention. The majority, however, provide at least a potential for intervention in order to slow or halt the progression of early CKD :

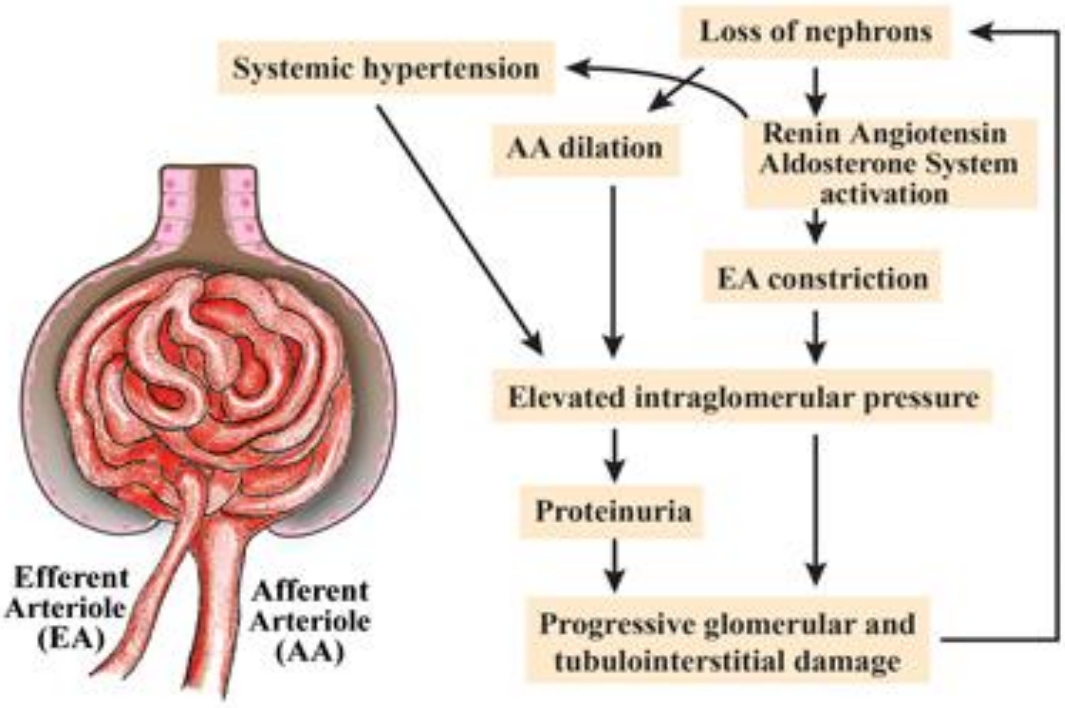
- High level proteinuria
- Severity of high blood pressure
- Poor glycemic control
- Type of nephropathy (Glomerulonephropathy& vascular nephropathy have more tendency to progress quickly).
- Intensity of tubulointerstitial damage in glomerulonephropathy.
- Smoking
- Exposure to industrial, environmental or medical nephrotoxic substances.
- Ethnic factors (A faster progression in blacks than in Caucasians).
- Genetic factors, such as: polymorphism of the different components
- The renin-angiotensin aldosterone system (controversial and marginal effect).
- Gender (controversial).

7.2 Pathophysiology of CKD progression:(89)

The progression of early CKD is mediated by a common pathophysiology resulting in glomerulosclerosis and tubulointerstitial injury. Many of the factors associated with provoking this renal injury, and thus progression of CKD are associated with, or caused by, chronic kidney disease itself. By targeting therapies to try to break this vicious circle we can aim to slow or halt progression.

Reduction of intra-glomerular hypertension/hyperfiltration, systemic hypertension and proteinuria, ideally by blocking the role of angiotensin II and attention to hyperlipidaemia, acidosis and phosphate balance with lifestyle modification where appropriate can have a beneficial impact upon the progression of CKD.

Figure 12 : Progression of Chronic Kidney Disease



ü Hypertension and progression of CKD:

ü Systemic hypertension is common in CKD and incidence increases with declining GFR [90]. Hypertension is due to a combination of sodium and water excess, activation of the renin–angiotensin–aldosterone system and sympathetic nervous system activation. Systemic hypertension is transmitted causing glomerular capillary hypertension and a subsequent accelerated decline in GFR as outlined above (intra-glomerular haemodynamic factors).

ü The kidneys also generate angiotensin II locally, independent of the systemic system, and acting via the angiotensin II type 1 receptor this has significant non-haemodynamic effects contributing to the development of tubulo-interstitial fibrosis by stimulating expression of cytokines and growth factors favouring fibrosis and recruitment of macrophages.[91] Thus, the use of angiotensin-converting enzyme (ACE) inhibitors performs a dual role, both lowering the glomerular capillary pressure and preventing the increase in pro-fibrotic cytokine expression.

ü Proteinuria and progression of CKD:

ü Proteinuria occurs as a result of glomerular capillary hypertension and varies directly with the intraglomerular pressure. The resulting damage to the permeability barrier in the glomerulus, a mechanism at least in part mediated by angiotensin II, leads to an excess of proteins reaching the lumen of the proximal tubules. Filtered proteins are taken up by the tubular cells by endocytosis and stimulate the abnormal production of cytokines which are released into the interstitium leading to macrophage and T lymphocyte migration, the proliferation of fibroblasts and increased ECM production: the familiar mechanisms of glomerulosclerosis and interstitial fibrosis[92]. Some specific proteins may play an enhanced role: transferrin is seen to accumulate

in the proximal tubular cells in proteinuric patients with CKD[93] and has been implicated in toxicity via lipid peroxidation and complement activation.

- Consistent with this role in pathophysiology, proteinuria is a strong predictor of clinical progression of CKD, the rapidity of progression is proportional to the severity of proteinuria.[94]

8. Complications of CKD :

Progression of CKD is associated with a number of serious complications, including increased incidence of cardiovascular disease, hyperlipidemia, anemia and metabolic bone disease. CKD patients should be assessed for the presence of these complications and receive optimal treatment to reduce their morbidity and mortality. (95)

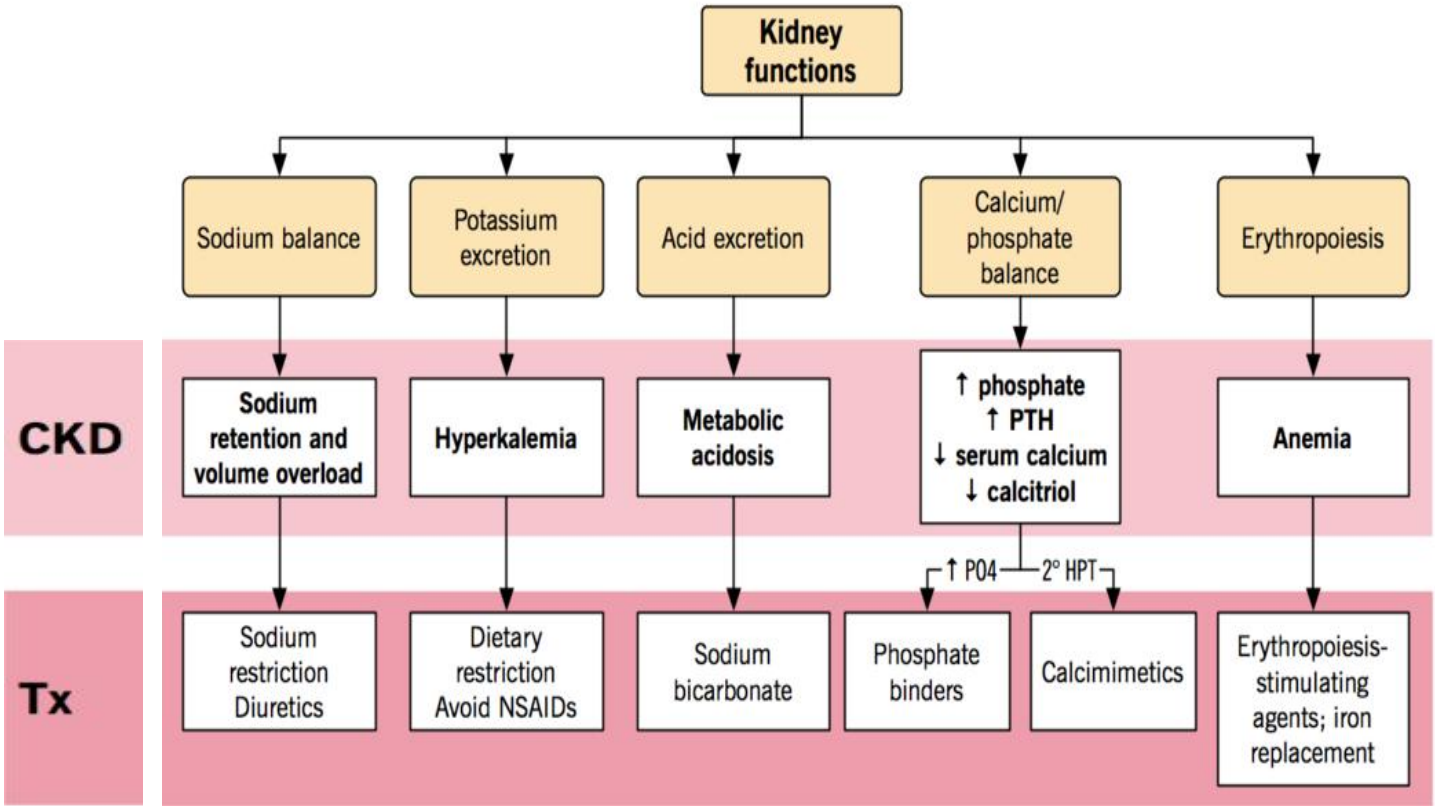


Figure 13 : Complications of CKD

8.1 Anemia:

A normochromic, normocytic anemia usually accompanies progressive CKD, (96) and the overall prevalence of CKD-associated anemia is approximately 50% (97). Although anemia may be diagnosed in patients at any stage of CKD, there is a strong correlation between the prevalence of anemia and the severity of CKD. One quarter of stage 1 CKD patients, half of those stratified to CKD stages 2, 3, and 4 and three quarters of CKD patients starting dialysis suffer from anemia(98).

Therefore, primary care providers play an important role in diagnosing and managing anemia in CKD patients.

While anemia in CKD can result from multiple mechanisms (iron, folate, or vitamin B12 deficiency; gastrointestinal bleeding; severe hyperparathyroidism, systemic inflammation, and shortened red blood cell survival), decreased erythropoietin synthesis is the most important and specific etiology causing CKD-associated anemia. Erythropoietin is a glycoprotein secreted by the kidney interstitial fibroblasts[99] and is essential for the growth and differentiation of red blood cells in the bone marrow. In CKD, tubular atrophy generates tubulointerstitial fibrosis, which compromises renal erythropoietin synthetic capacity and results in anemia.

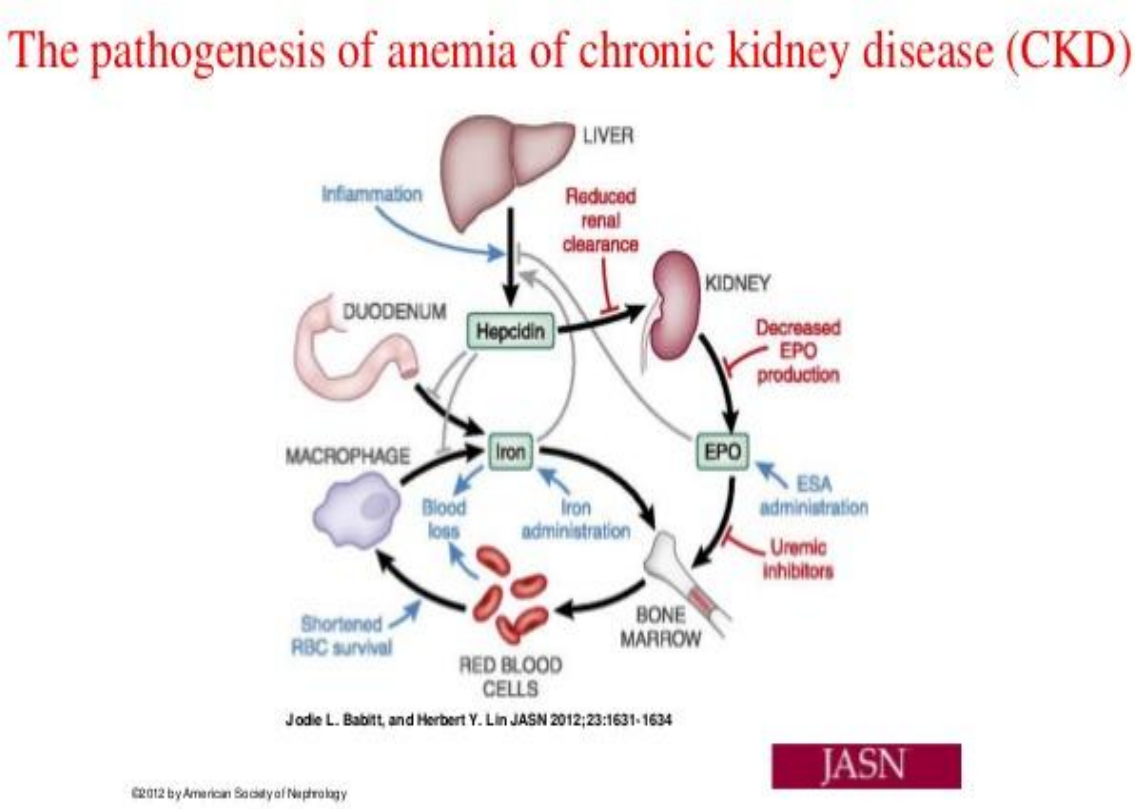
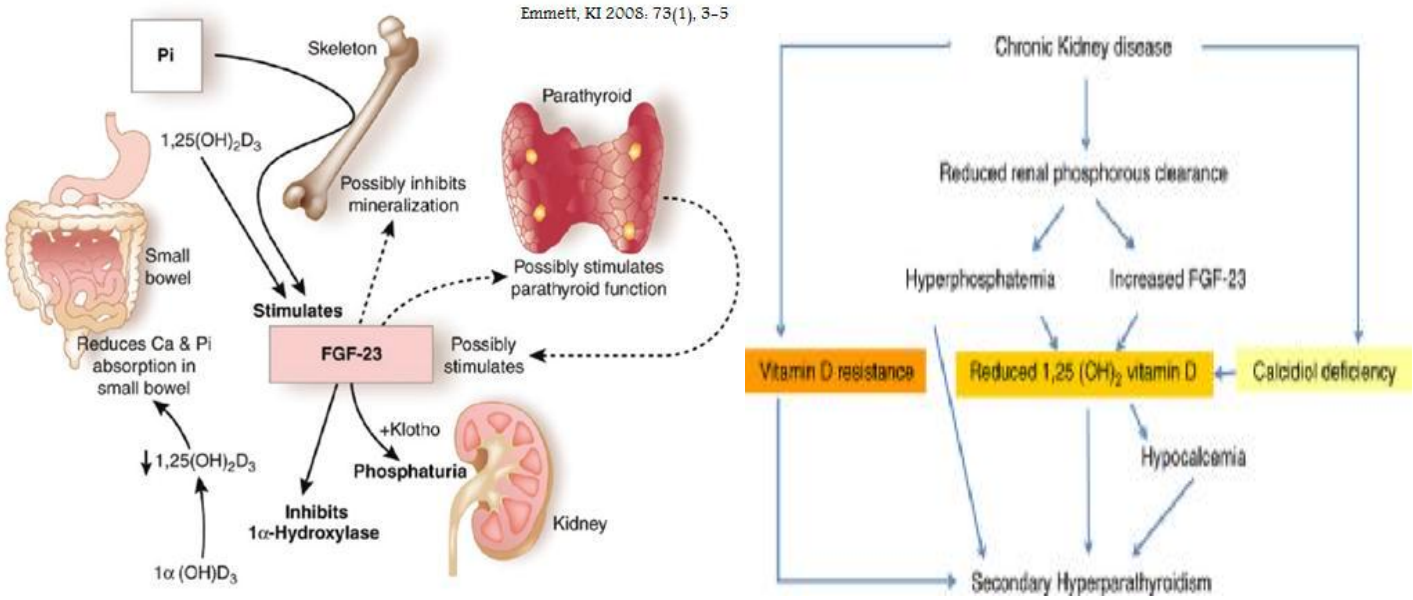


Figure 14 : chronic kidney disease related Anemia

8.2 Mineral and bone disorders: (95)

Renal osteodystrophy is the spectrum of histological changes, which occur in bone architecture of patients with CKD. The kidney is the primary site for phosphate excretion and 1-α-hydroxylation of vitamin D. CKD patients develop hyperphosphatemia as a result of inadequate 1, 25 dihydroxy-vitamin D levels that reflect reduced synthesis from parenchymal scarring. In addition, renal phosphate excretion is reduced. Together both processes cause, serum calcium levels to fall resulting in increased secretion of parathyroid hormone (secondary hyperparathyroidism). Parathyroid hormone has a phosphaturic effect. It also increases the calcium levels by increasing bone resorption and promoting 1-α-hydroxylation of 25-hydroxy vitamin D synthesized by the liver (limited effect because of reduced kidney reserve from scarring).

Changes in bone architecture can be caused by either a high bone turnover state or a low bone turnover state. Four types of bone phenotypes (renal osteodystrophy) can be diagnosed in CKD patients: osteitis fibrosacystica (high bone turnover with secondary hyperparathyroidism), osteomalacia (low bone turnover and inadequate mineralization, primarily related to diminished vitamin D synthesis), a dynamic bone disorder (low bone turnover from excessive suppression of the parathyroid glands), and mixed osteodystrophy (with elements of both high and low bone turnover). (Figure 14)



a b

Figure 15 : Mineral & bone disorders in chronic kidney disease : Role of Vitamin D (figure 15a) and FGF-23 (figure 15b)

8.3 Cardio vascular risk:

The increased cardiovascular risk associated with end stage renal disease has been well established, and estimated cardiovascular mortality rates are ten to one hundred fold higher among dialysis patients than age- and sex-matched individuals in the general population.(100) Many traditional cardiovascular risk factors, documented in the general population, contribute to cardiovascular risk in CKD patients. In fact, many Framingham risk factors are more prevalent among individuals with CKD than those with normal renal function. However, non-traditional risk factors, specific to CKD patients, notably hypertension, diabetes, anemia, proteinuria and inflammation are considered the most incriminated underlying causes that contribute to the burden of cardiovascular disease(95).

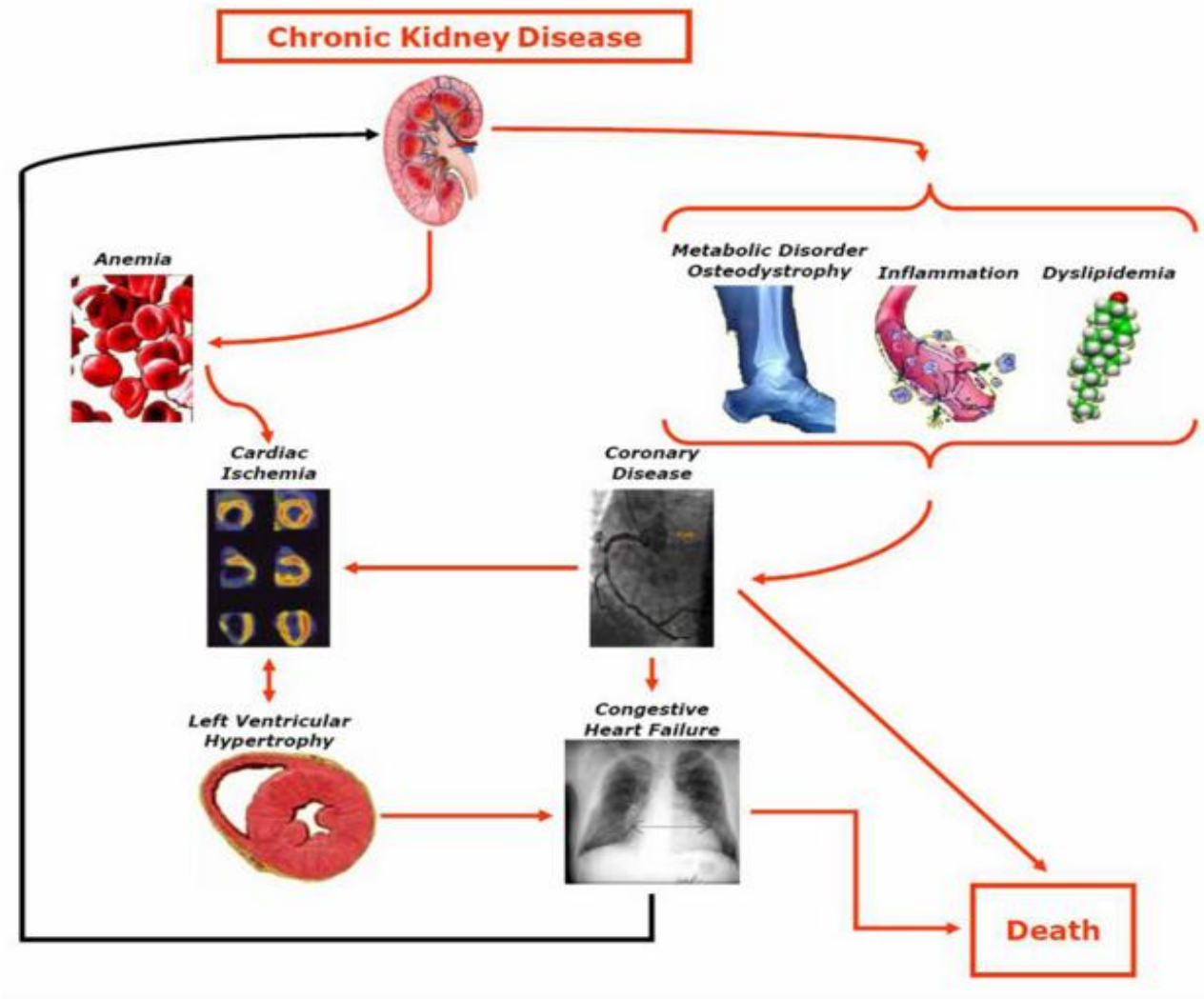


Figure 16 :Interplay of processes secondary to chronic kidney disease leading to cardiovascular disease and death.

Red arrows: Pathogenetic pathways;

Black arrow: Feedback loop; kidney disease worsened by heart failure.

8.4 Dyslipidemia:[101]

Dyslipidemia is a major risk factor for cardiovascular morbidity and mortality and is common among patients with CKD. Lipid profiles vary widely in these patients, reflecting the level of kidney function and the degree of proteinuria. In general, the prevalence of hyperlipidemia increases as renal function declines, with the degree of hypertriglyceridemia and elevation of LDL cholesterol being proportional to the severity of renal impairment.

Several factors contribute to the development dyslipidemia associated with chronic renal impairment. Patients with CKD have a reduction in the activity of lipoprotein lipase and hepatic triglyceride lipase. This interferes with uptake of triglyceride-rich, apolipoproteinB-containing lipoproteins by the liver and in peripheral tissue, yielding increased circulation of these atherogenic lipoproteins. Hypercholesterolemia in nephrotic syndrome is thought to be due to increased production and decreased catabolism of lipoproteins. The degree of lipoprotein abnormality is roughly proportional to the amount of proteinuria and inversely proportional to serum albumin levels.

8.5 Nutritional issues:[95]

As patients progress through the stages of CKD, nutritional requirements are altered and metabolism of protein, water, salt, potassium, and phosphorous are affected [102]. These changes lead to ineffective energy generation despite adequate intake of protein and carbohydrate substrates. In more extreme manifestations, these alterations in nutrient utilization cause “uremic malnutrition,” a syndrome that is distinct from malnutrition caused by inadequate nutrient intake. Both inadequate nutrient intake and ineffective nutrient utilization can contribute to nutritional disorders in CKD patients and we will not distinguish between these etiologies in our discussion. The association between uremic malnutrition and outcomes in the early

stages of CKD has not been investigated. However, there is adequate evidence to suggest that a poor predialysis, nutritional status increases patient morbidity and mortality after initiation of renal replacement therapy [103]. Maintenance of neutral nitrogen balance is important for preservation of nutritional health in patients with chronic renal impairment. Treatment goals in this setting should be to establish and maintain optimal nutritional status, minimize uremic symptoms and signs as renal impairment declines, and to establish a nutritional plan that is acceptable to the patient. To accomplish these goals, involvement of a dietician in the care of these patients is often necessary.

9. Management of CKD:

- The leading purpose of management in all stages is preventing progression and complications of CKD . The individual's ability to manage his/her health situation and adherence to medication and diet prescriptions are crucial in the efforts to slow down the disease progress and prevent complications.

9.1 Conservative management:[104-110]

In early CKD stages (1-3 stages), management is based on :

a. Control of CKD risk factors:

- Tight blood pressure control to target values per current guidelines:
  - Use of renin-angiotensin system (RAS) blockers among patients with diabetic kidney disease (DKD) and proteinuria.
  - Use of angiotensin-converting enzyme inhibitors (ACEIs) or angiotensin-receptor blockers (ARBs) in patients with proteinuria.
  - Control of HBP Reduces damage due to the end organ effects of hypertension on the kidney as well as the heart. In fact, Angiotensin-converting enzyme inhibitors (ACEI) and angiotensin II receptor

blockers(ARBs), block the effects of angiotensin II on (i) sodium and fluid retention, (ii) vasoconstriction, (iii) stimulating ADH release, (iv) stimulating aldosterone release, and (v) inducing a sympathetic response. ACEIs and ARBs also slow down progression of proteinuria in patients with diabetic CKD.

- Reduction of proteinuria
- Aggressive glycemic control (target hemoglobin A1c [HbA1C] < 7%):
  - Tight glucose management slows the progression of vascular and heart disease.
- Treatment of hyperlipidemia to target levels per current guidelines.
- And other risk factors, such as life style issues like over/underweight, physical inactivity and smoking.

b. Treatment of the underlying condition if possible

c. Dietetics:

- Mixed evidence exists whether dietary protein restriction is beneficial in slowing disease progression.
- Proteins affect the renal hemodynamics, raising the GFR, in hypothesized 2 ways.
  - *Hormonal effects* – proteins cause secretion of glucagon, IGF-1 and kinins, all of which have been shown to raise the GFR.
  - *Tubuloglomerular effects* – high amino acid (AA) filtration leads to increased AA and hence the sodium uptake in the proximal convoluted tubule. A decreased sodium delivery to the distal convoluted tubule leads to the rennin-angiotensin system activation via the macula densa and these work to raise the GFR (mechanisms above)

- Controlling hyperphosphatemia: Protein restriction also limits phosphorus consumption. Hyperphosphatemia plays a major role in the progression of renal osteodystrophy. Phosphate binders are used to reduce phosphate absorption through the GI tract.

d. Avoidance of nephrotoxins, including intravenous (IV) radiocontrast media, non-steroidal anti-inflammatory agents (NSAIDs), and aminoglycosides

e. Treating Pathologic Manifestations of Chronic Kidney Disease:

- Anemia: When the hemoglobin level is below 10 g/dL, treat with an erythropoiesis-stimulating agent (ESA) such as epoetinalfa or darbepoetinalfa (previously, peginesatide was also considered an option for anemia in CKD, but this agent was withdrawn from the market in February 2013 due to serious hypersensitivity reactions [68]); caution should be exercised in patients with malignancy.
- Hyperphosphatemia: Treat with dietary phosphate binders and dietary phosphate restriction.
- Hypocalcemia: Treat with calcium supplements with or without calcitriol.
- Hyperparathyroidism: Treat with calcitriol, vitamin D analogues, or calcimimetics
- Volume overload: Treat with loop diuretics or ultrafiltration.
- Metabolic acidosis: Treat with oral alkali supplementation
- Uremic manifestations: Treat with long-term renal replacement therapy (hemodialysis, peritoneal dialysis, or renal transplantation)
- Cardiovascular complications: Treat as appropriate
- Growth failure in children: Treat with growth hormone

9.2 Renal replacement therapy (RRT):

- In CKD 4-5, pharmacological treatment is usually intensified. Additionally this treatment is supplemented with dietary changes and occasionally fluid restrictions. Interventions to prepare for RRT (i.e. patient education and decision of RRT alternative), access surgery and/or preparing for kidney transplantation are commenced.
- Indications of RRT:

  RRT is usually started when the patient reaches end-stage renal disease (ESRD) at GFR approximately 5–10 ml/min/1.73/m^2. Indications for renal replacement therapy in patients with chronic kidney disease (CKD) include the following:

  - Severe metabolic acidosis
  - Hyperkalemia
  - Pericarditis
  - Encephalopathy
  - Intractable volume over load
  - Failure to thrive and malnutrition
  - Peripheral neuropathy
  - Intractable gastrointestinal symptoms
  - In asymptomatic adult patients, a glomerular filtration rate (GFR) of 5-9 mL/min/1.73 m^2, [2] irrespective of the cause of the CKD or the presence of absence of other comorbidities.
- Preparation of patients for RRT :

  Timely planning for long-term RRT consider the following :

- Early patient education regarding natural disease progression, different dialytic modalities, renal transplantation, and option to refuse or discontinue chronic dialysis
- Timely placement of permanent vascular access (arrange for surgical creation of primary arteriovenous fistula, if possible, and preferably at least 6 mo in advance of the anticipated date of dialysis for patients in whom transplantation is not imminent)
- Timely elective peritoneal dialysis catheter insertion
- Timely referral for renal transplantation

ü RRT options are peritoneal dialysis (PD), haemodialysis (HD) or kidney transplant, which all imply lifelong treatment. However, Some individuals – often elderly patients with multiple comorbid conditions – opt for conservative management, aiming at preservation, preventing and managing complications and palliation.

a.Hemodialysis (HD):(111)(112)

ü HD is the most used renal replacement therapy in Morocco. Its main advantage in comparison with peritoneal dialysis is giving a better technical survival and a reduced hospitalization rates.

ü HD usually involves treatment sessions at least thrice weekly, carried out either at outpatient units or through self-care at home.

ü Contraindications of HD include:

1. Serious bleeding or anemia.
2. Severe hypotension or shock.
3. Serious heart or brain complications, i.e. obvious cardiomegaly with insufficiency of heart function, serious arrhythmia, severe hypertension or brain blood vessel disease.

4. End-stage uremia with serious irreversible complications.
5. uncontrolled diabetes.
6. Serious infection.
7. Have malignant disease such as cancer at the same time.
8. Still within the 3 days after a big operation.
9. Old high-risk patients, non-cooperative mental disorder patients, infant and children

ü Hemodialysis principal:

The principle of hemodialysis is the same as other methods of dialysis; it involves diffusion of solutes across a semipermeable membrane. Hemodialysis utilizes counter current flow, where the dialysate is flowing in the opposite direction to blood flow in the extracorporeal circuit. Counter-current flow maintains the concentration gradient across the membrane at a maximum and increases the efficiency of the dialysis.

Fluid removal (ultrafiltration) is achieved by altering the hydrostatic pressure of the dialysate compartment, causing free water and some dissolved solutes to move across the membrane along a created pressure gradient.

The dialysis solution that is used may be a sterilized solution of mineral ions or comply with British Pharmacopoeia. Urea and other waste products, potassium, and phosphate diffuse into the dialysis solution. However, concentrations of sodium and chloride are similar to those of normal plasma to prevent loss. Sodium bicarbonate is added in a higher concentration than plasma to correct blood acidity. A small amount of glucose is also commonly used.

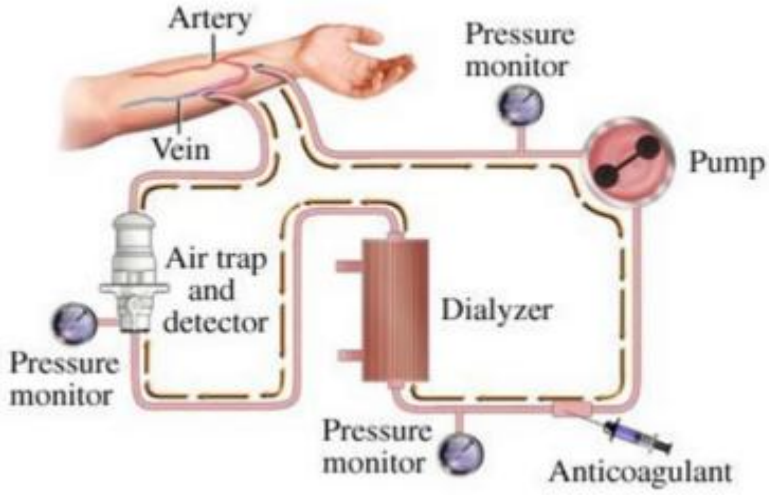


Figure 17: principal of hemodialysis(113).

b. Peritoneal dialysis (PD):(114)

- Peritoneal dialysis (PD) is an alternative treatment to haemodialysis that can be carried out at home, at work, or on trips, but requires careful supervision. It gives patients more control. However, they need to work closely with the health care team including the nephrologist, dialysis nurse, dialysis technician, dietitian and social worker.
- Inn Morocco, peritoneal dialysis is rarely practiced. Of the 2657 prevalent cases who are on dialysis, only 7 cases (0.3%) are on peritoneal dialysis in the 4 regions where the register is located, means a prevalence of 0.7 pmp.(115)
- Principal of PD :

  A special sterile fluid is introduced into the abdomen through a permanent tube that is placed in the peritoneal cavity. The fluid circulates through abdomen to draw impurities from surrounding blood vessels in the peritoneum, which is then drained from the body.(figure16)

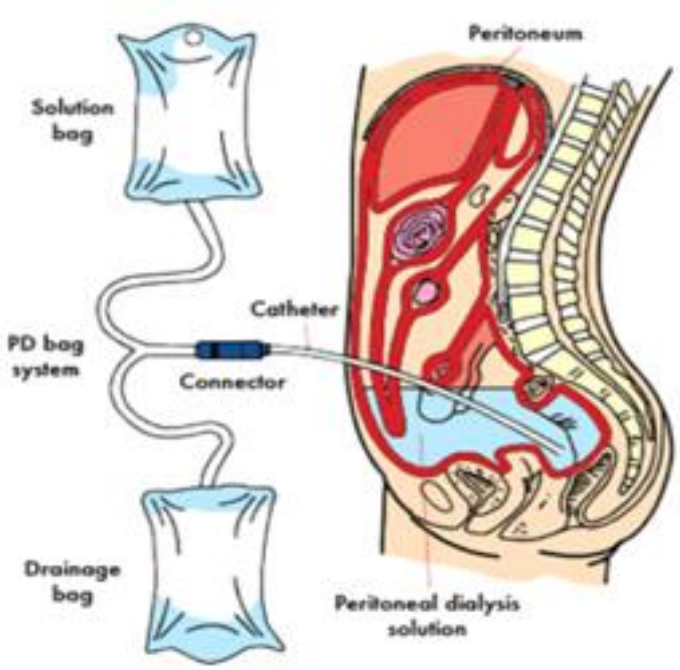


Figure 18: Peritoneal dialysis principal

A PD catheter is inserted permanently at the abdomen to allow filling and draining of about two litres of PD solution into and out of the peritoneum or abdominal cavity, which is surrounded by the peritoneal membrane. The peritoneal membrane then filters waste and fluids from the blood into the solution.(figure 17)

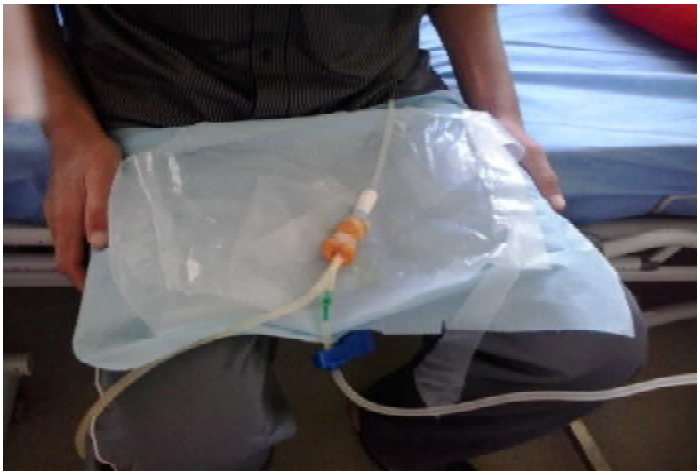


Figure 19: PD catheter

The PD solution is allowed to remain in the abdomen for four to six hours before it is drained and replaced with fresh PD solution. The replacing of fresh PD solution with the used PD solution is called an exchange. Each exchange takes about 30 minutes. PD patients perform an average of four exchanges per day. Different types of PD have different schedules of daily exchanges.

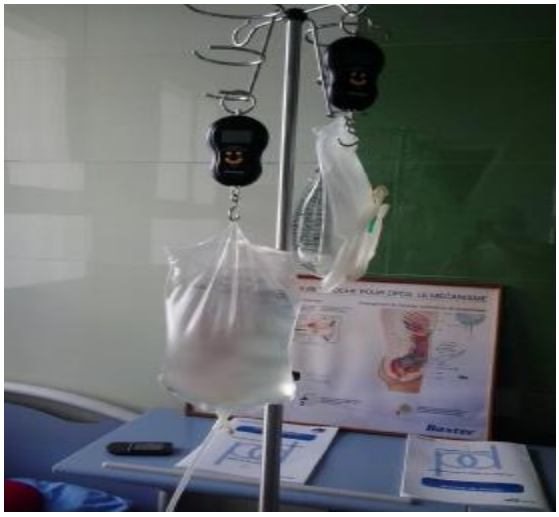


Figure 20: PD solution

ü Types of PD :

1) Continuous ambulatory peritoneal dialysis: Unlike Haemodialysis, patients do not need a machine for CAPD. They need gravity to fill and empty their abdomen. The doctor will prescribe the number of exchanges a patient needs, typically three or four exchanges during the day and one evening exchange with a long overnight dwell time while one sleeps. As the word “ambulatory” suggests, the patient can walk around with the dialysis solution in the abdomen.

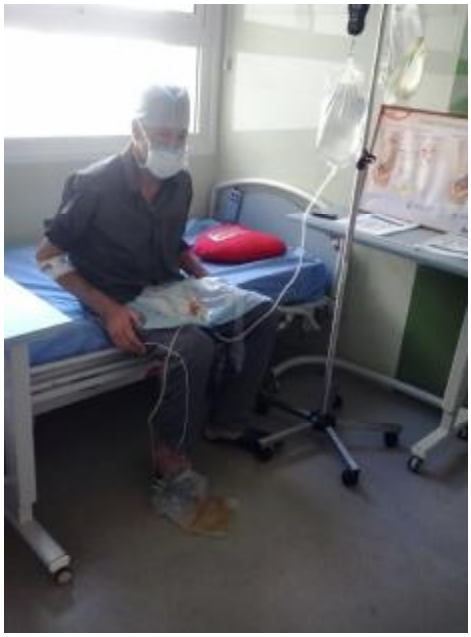


Figure 21: Continuous ambulatory peritoneal dialysis (CAPD)

2) Automated peritoneal dialysis: An alternative to CAPD is Automated Peritoneal Dialysis (APD) where a machine called a cycler will change the dialysate solution during the night, usually while patients are asleep. This means that patients have to be attached to the machine for 8-10 hours.

Figure 22 : Automated peritoneal dialysis (CAPD)

ü Benefits of PD :

Painless and No Needling

- Unlike HD, no vascular access or needling is required for PD, hence PD is a painless procedure.
- Vascular access-related complications are one of the common causes of hospitalisation in haemodialysis patients.

Home-based Therapy

- Patients carry out treatment themselves in the comfort of their own homes.
- No need to travel to the dialysis centre for treatment and not restricted to dialysis centres's schedule.
- More flexibility to better fit dialysis into their lifestyle.
- Patients take charge their own treatment plan and advice given by the PD care team.

Gentler and works more like the natural kidney

- PD mimics the function of real kidneys more as the constant presence of the PD solution in the abdominal cavity allows waste products and excess water from the blood to be removed continuously.
- The non-intermittent nature of PD makes it a gentler treatment. Patients have lesser food restrictions and experience lesser side effects

c.Renal transplant:[116]

ü Kidney transplant or renal transplant is the process where a kidney is surgically removed from a donor and implanted into the patient. The patient may receive a kidney from a living-donor, or from a recently deceased person, known as a cadaveric donor or deceased-donor.

- It is estimated that 250 is the number of kidney transplanted patients in Morocco so far. Moreover, only 1.2% of patients on dialysis are on the waiting list of kidney transplant, having as reason of non registration, a contraindication against the transplant in 14.2% of cases, or refusing the transplant in 17.3% of cases, and in 65% of cases, for other non explicit reasons.(115)
- Kidney transplant would be the best treatment for End-Stage Renal Disease (ESRD). However, the waiting list for kidney transplant is long. According to NKF, there are more than 400 individuals on the waiting list for cadaver renal transplant, to date. And the average waiting time for renal transplant is 9 years.

9.3 Nutritional management in CKD:

9.3.1 Nutritional guidelines in normal people:

Ø Energy needs: (117)(118)(119)

- Energy is not a nutrient but is required in the body for metabolic processes, physiological functions, muscular activity, heat production, growth and synthesis of new tissues. It is released from food components by oxidation.
- Three macronutrients form the main source of energy;

- Carbohydrates: which provide the major part of daily energy requirements with 50%,of which free sugars have to represent no more than 5%.
- Fats and oils: Constitute 35%of needs, where no more than 11% are provided by saturated fat.
- Then proteins with only15% of daily needs.

- In other words, the average amount of energy released ranges from approximately 16.7 kJ/g for carbohydrates or protein and 37.7 kJ/g for fats.
- Several factors define our energy needs, especially age, weight and physical activity. But other influences are also concerned :

- Sex – males release energy from their food 5–7% faster than females.
- Size – the more surface area a person has, the greater is the energy needs.
- Body composition – the higher the ratio of lean tissue to fat tissue, the higher is the need.
- Thyroid hormone – this hormone stimulates resting metabolism, then more enery is required
- Other factors – fever, certain drugs like caffeine, pregnancy, lactation and emotions all increase our energy needs.

- The following tables show the estimated energy requirements in both children and adults, according to the British Nutrition Foundation (BNF).

Table 6: Estimated average energy requirements for childeren

	Males		*Females*	
Age (yrs)	**MJ/d**	**kcal**	**MJ/d**	**kcal**
4	5.8	1386	5.4	1291
5	6.2	1482	5.7	1362
6	6.6	1577	6.2	1482
7	6.9	1649	6.4	1530
8	7.3	1745	6.8	1625
9	7.7	1840	7.2	1721
10	8.5	2032	8.1	1936
11	8.9	2127	8.5	2032
12	9.4	2247	8.8	2103
13	10.1	2414	9.3	2223
14	11.0	2629	9.8	2342
15	11.8	2820	10.0	2390
16	12.4	2964	10.1	2414
17	12.9	3083	10.3	2462
18	13.2	3155	10.3	2462

Table 7: Estimated average energy requirements for adults

	Males		*Females*	
Age (yrs)	**MJ/d**	**kcal**	**MJ/d**	**kcal**
19-24	11.6	2772	9.1	2175
25-34	11.5	2749	9.1	2175
35-44	11.0	2629	8.8	2103
45-54	10.8	2581	8.8	2103
55-64	10.8	2581	8.7	2079
65-74	9.8	2342	7.7	1912
75+	9.6	2294	8.7	1840

Ø Nutrients needs:

- The World health organization (WHO) and Food & Agriculture organization (FAO), arrange in this group over twenty essential nutrients, that comprise the basis of all human nutrition. These nutrients include protein, vitamins (A, B, C, D, E, K, B12...) and ions (Sodium, potassium, calcium...). As these micronutrients are present in very different ways in different food categories, covering their needs is a major reason for advocating a varied diet.(120)
- Proteins: (118)

ü In Adults:

- The Reference Nutrient Intake (RNI) is set at 0.75g of protein per kilogram bodyweight per day in adults.
- Protein requirements increase in pregnancy (an additional 6g/d) and lactation (an additional 11g/d 0-6m and 8g 6+ months).

ü In children:

Table 8: Reference proteins intake in children

Age group	RNI per day (g)
0-3m	12.5
4-6m	12.7
7-9m	13.7
10-12m	14.9
1-3y	14.5
4-6y	19.7
7-10y	28.3

- Sodium:[118]The target salt intakes set for adults and children do not represent ideal or optimum consumption levels, but achievable population goals.

Table 9: Reference salt intake in children

Age group	Recommended intake per day (g)
2-5 years	15g / day
5-11 years	20g / day
11-16 years	25g / day
17 years and over	30g / day

Table 10: Reference salt intake in adults

Age group	Maximum salt intake per day (g)
0-6 months	<1g / day
6-12 months	1g / day
1-3 years	2g / day
4-6 years	3g / day
7-10 years	5g / day
11 years and above	6g / day

- calcium:[121][122]

- The UK reference nutrient intake (RNI) for calcium for adults aged over 19 years is 700mg/day; while according the American Scientific Committee for Food, the average daily calcium requirement is 550mg/kg, with 400mg as a lowest intake.
- Requirements are higher during childhood, adolescence and during lactation, indeed Between the ages of 1 and 10, the average daily calcium retention needed for skeletal growth has been estimated to rise from 70 to 150 mg/d.[123]

Table 11: Reference nutrient intakes for minerals [118]

Age	Calcium	Phosphorus[1]	Magnesium	Sodium	Potassium
	mg/d	mg/d	mg/d	mg/d[2]	mg/d[3]
0-3 months	525	400	55	210	800
4-6 months	525	400	60	280	850
7-9 months	525	400	75	320	700
10-12 months	525	400	80	350	700
1-3 years	350	270	85	500	800
4-6 years	450	350	120	700	1100
7-10 years	550	450	200	1200	2000
Males					
11-14 years	1000	775	280	1600	3100
15-18 years	1000	775	300	1600	3500
19-50 years	700	550	300	1600	3500
50+ years	700	550	300	1600	3500
Females					
11-14 years	800	625	280	1600	3100
15-18 years	800	625	300	1600	3500
19-50 years	700	550	270	1600	3500
50+ years	700	550	270	1600	3500
Pregnancy	*	*	*	*	*
Lactation:					
0-4 months	+ 550	+ 440	+ 50	*	*
4+ months	+ 550	+ 440	+ 50	*	*

9.3.2 Nutritional guidelines in patients with CKD:

Ø Nutritional recommendations in Non-dialyzed patients:

- Before reaching the End Stage renal Disease (ESRD) and resorting to Renal Replacement therapy (RRT), the nutritional management aims particularly to reduce the daily intakes of proteins, phosphorus and potassium, in order to decrease the risk of acidosis, hyperparathyroidism, insulin resistance and cardiovascular disease.

Energy & proteins:[124][125]

- The attempts to limit protein intake exposes to the risk of malnutrition, comorbidity and mortality even. This makes dietary protein prescription in chronic kidney disease, complicated by potential conflict between goals to slow the progression of kidney disease and preserve protein nutritional status.
- Thus, according to NKF, a recommended daily intake for proteins of 0.75 g/kg appears reasonable in patients with GFR >30 mL/min/1.73 m^2 (CKD Stages 1–3).
- While in CKD Stages 4-5 patients who are not undergoing maintenance dialysis, the institution of a planned low-protein diet providing 0.60 g protein/kg/d should be considered.
- For individuals who will not accept such a diet or who are unable to maintain adequate dietary energy intake with such a diet, an intake of up to 0.75 g protein/kg/d may be prescribed.
- The K/DOQI Nutrition Guidelines, recommends also for Non-dialyzed individuals a Dietary Energy Intake (DEI) of 35 kcal/kg/d for those who are younger than 60 years old and 30-35kcal/kg/d for individuals who are 60 years of age or older.

- The nutritional status of non dialyzed patients with chronic kidney disease should be monitored at regular intervals: every 1 to 3 months for patients with GFR <30 mL/min/1.73 m² (CKD Stages 4 and 5) and every 6 to 12 months for patients with GFR 30 to 59 mL/min/1.73 m² (CKD Stage 3).

Table12 :Recommendations for protein supply in adult patients with CRF (g/kgBW/day).[125]

	ESPEN	NKF
GFR = 25–70 ml/min	0.55–0.60* (2/3 HBV)	—
GFR<25 ml/min	0.55–0.60 (2/3 HBV) or 0.28+EAA or EAA+KA	0.60 or 0.75 (intolerance or inadequate energy intake)

ESPEN, European Society for Clinical Nutrition and Metabolism; NKF, National Kidney Foundation; EAA, essential amino acids; GFR, glomerular filtration rate; HBV, high biological value; KA, ketoanalogues.

Micronutrients intakes:

- Sodium: Clinical and experimental studies clearly show that sodium handling by the kidney is altered in CKD, and that sodium retention has a major role in hypertension in CKD. The primary mechanism appears to be expansion in extracellular fluid (ECF) volume. Dietary sodium restriction is recommended to reduce ECF volume expansion and lower blood pressure. The national kidney Foundation recommended that most individuals with CKD should reduce sodium intake to less than 2.5 g/d. Further reduction in sodium intake to <50 mmol/d (<1.2 g/d) might lower blood pressure further, but may be more difficult to achieve.[125-128]
- Potassium: As regarding potassium intakes, for CKD stages 4-5, the dietary potassium intake recommended by the ESPEN guidelines is 1500-2000mg/day. While in CKD stages 1-3, the daily requirements of potassium must be matched to laboratory values.[126]

- Calcium: The recommended calcium intakes in stages 3-5 (predialysis) is <2000 mg/day(including calcium from phosphate binders).(126)(127)
- Phosphorus: In CKD stage 3-5: 600/800 – 1,000 mg/day of Phosphorus, is the recommended daily intake, if levels of phosphate or parathyroid hormone are elevated.(125-128)

Table 13 : Mineral requirements in patients with CRF.(125)

Phosphate	600–1000 mg/d*
Potassium	1500–2000 mg / d†
Sodium	1.8–2.5 g/d‡
Fluid	not limited‡

*Depending on physical activity, lean body mass, age, gender, degree of malnutrition, etc.
†Individual requirements can differ considerably.
‡Such a large span of protein requirement depends on degree of renal insufficiency, dietary habits, energy intake, rate of progression or renal failure, etc.

Ø Nutritional recommendations in dialyzed patients:(129)

- The nutritional support of the dialysis patient aims to control the intake of some nutrients (phosphorus and potassium) and reduce the accumulation of metabolic wastes (urea) between dialysis sessions. This must be done by taking into account the dietary guidelines for the general population for aspects not directly related to renal disease and the preferences of the patient. The specific aims of nutritional support are:

- To reduce the accumulation of metabolic wastes, fluids and electrolytes;
- To prevent metabolic complications of CKD;
- To replace nutrients lost with dialysis;
- To promote a satisfactory nutritional status

Energy & proteins:

The dialysis modality affects the nutritional needs of CKD patients. For instance, PD patients have to decrease their energy intake because of the absorption of glucose from the dialysate. On the other hand, their protein requirements may be higher because of protein losses through the peritoneal membrane. The dietary demands of patients on HD are more complex than PD patients and this is confirmed throughout this paper, which concentrates more on the needs of HD patients.(129)

Table 14: Estimated energy requirements for dialysis patients

	Haemodialysis	Peritoneal dialysis
DOQI	1.2 g/kg IBW/day	1.2–1.3 g/kg IBW/day
	≥50% HBV	≥50% HBV
ESPEN	1.2 g/kg IBW/day	1.2–1.5 g/kg IBW/day
	≥50% HBV	≥50% HBV
		Greater intake if peritonitis
EBPG	At least 1.1 g/kg IBW/day	
	Balanced intake of high quality animal protein and vegetable protein source	
EDTNA/ERCA	1.1–1.2 g/kg IBW/day	1.0–1.2 g/kg IBW/day
		1.5 g/kg IBW/day if peritonitis
	≥50% HBV	≥50% HBV

Table 15:Estimated protein requirements in dialysis patients

	Haemodialysis	Peritoneal dialysis
DOQI	35 kcal/kg BW/day	35 kcal/kg BW/day
	30–35 kcal/kg BW/day if age ≥ 60 years	30–35 kcal/kg BW/day if age ≥ 60 years
		Consider energy provided by dialysate
ESPEN	35 kcal/kg IBW/day	35 kcal/kg IBW/day
		Fats 30–40% of total energy supply; complex carbohydrates 25–40%; simple sugars restricted
		Glucose absorption must be taken into account
EBPG	30–40 kcal/kg IBW/day	
	Adjusted to age, gender and physical activity level	
EDTNA/ERCA	35 kcal/kg IBW/day	35 kcal/kg IBW/day
	30 kcal/kg IBW/day in the elderly and patients with reduced activity	30 kcal/kg IBW/day in the elderly and patients with reduced activity
		Including calories from peritoneal absorption of glucose

Micronutrients intake:

- Phosphorus: Hyperphosphataemia is common in stage advanced CKD patients. A diet with the suggested quantity of protein for a CKD patient carries about 10–13 mg of phosphorus per gram of protein while a single dialysis session can remove up to 800 mg of phosphorous. The ensuing hyperphosphataemia is implicated in hyperparathyroidism, mineral bone disease and cardiovascular disease (NKF 2003). It should be noted that, while increasing protein intake will slightly increase serum phosphate. Table 13 gives the suggested dietary intake of phosphorus for dialyzed CKD patients.(129)

Table 16: Suggested phosphorus intake for the dialysis(130) patient.(130)

	Haemodialysis	Peritoneal dialysis
DOQI (NKF 2003)	Restricted to 800–1000 mg/day if serum phosphorus level >5.5 mg/dL	Restricted to 800–1000 mg/day if serum phosphorus level > 5.5 mg/dL
ESPEN	17 mg kg IBW	17 mg kg IBW
EBPG	800–1000 mg/day	–
EDTNA/ERCA	1000–1400 mg/day	1000–1400 mg/day

- Potassium: Control of dietary potassium is important to prevent hyperkalaemia between dialysis sessions. The kidney excretes 90% of dietary potassium and the intestinal excretion of potassium is increased during CKD, as a compensatory mechanism. For this reason, constipation may favourhyperkalaemia while diarrhea may be responsible for hypokalaemia because of potassium loss. Dialytic efficiency is central to the maintenance of an acceptable potassium level and the use of a potassium enriched dialysate should be avoided, if not strictly necessary. Table 14 gives the suggested dietary intake of potassium for CKD dialyzed patients.(129)

Table 17: Suggested potassium intake for the dialysis(131) *patient.* *(131)*

	Haemodialysis	Peritoneal dialysis
DOQI	N/A	N/A
ESPEN	<1 mEq/kg /day	<1 mEq/kg /day
EBPG	<1 mEq/kg IBW/day or 50–70 mmol/day	
EDTNA/ERCA	2000–2500 mg/day	2000–2500 mg/day

- Sodium & Fluid: In stage 5 CKD there is a substantial decrease of sodium excretion, which may be responsible for an increase in extracellular water. Such expansion is responsible for hypertension while a too rapid removal of sodium during dialysis is associated with hypotension and arrhythmias. As noted above, CKD patients should not use a salt substitute as it may increase the risk of hyperkalaemia.(129)
- The European Best Practice Guidelines (EBPG), recommends 500-1000mL/day as a daily requirement of fluid, for hemodialyzed patients, while other foundations recommend the following needs (Table 16).

Table 18: Suggested sodium intake for the dialysis patient.(131)

	Haemodialysis	Peritoneal dialysis
DOQI	N/A	N/A
ESPEN	60–100 mEq/day	60–100 mEq/day
EBPG	80–100 mEq/day	
EDTNA/ERCA	1800–2500 mg/day	1800–2500 mg/day

Table 19: Suggested fluid intake for the dialysis patient.(131)

	Haemodialysis	Peritoneal dialysis
DOQI	N/A	N/A
ESPEN	500–800 mL/day + daily urine output (taking into account the fluid content of foods)	500–800 mL/day + residual urinary volume (taking into account the fluid content of foods)
EBPG	Intradialytic weight gain < 4–4.5% of dry body weight	
EDTNA/ERCA	500 mL + daily urine output – includes only foods that are liquid at room temperature and those with high fluid content	800 mL + daily urine output – includes only foods that are liquid at room temperature and those with high fluid content

- Calcium: According to the European Best practice Guidelines (EBPG), the dietary sodium intake in patients on maintenance dialysis must be less than 2000mg/day, including calcium from phosphate binders. (132)

Ø Nutritional recommendations in diabetic patients:(133)

- Nutritional management for people with diabetes has traditionally focused on blood glucose control. However, dietary protein intake at all stages of CKD appears to have an important impact in this population.
- If dietary protein is limited, adequate caloric intake must be maintained by increasing calories from carbohydrates and/or fats. Competing needs for nutritional management of hyperglycemia, hypertension, and dyslipidemia can make determination of appropriate protein intake challenging.
- Furthermore, the diet for diabetes and CKD should consider the qualitative, as well as the quantitative, aspects of proteins, carbohydrates, and fats.
- A dietary protein intake of 0.8 g/kg body weight per day, is the recommended dietary allowance (RDA) for this macronutrient, it is a level that has been achieved in studies of diabetes and CKD.
- If dietary protein intake is limited, an increase in carbohydrates and/or fats is required for adequate caloric intake. Increasing intake of omega-3 and monounsaturated fats may confer benefits on CKD.
- The following table show the varied RDA of protein and micronutrients in diabetics patients according to CKD stages:

Table 20:A Balanced Approach to Nutrition in CKD With Diabetes: Macronutrient Composition and Mineral Content

Nutrient	Stage of CKD		
	1-2	1-4	3-4
Sodium (g/d)		<2.3	
Total fat* (% of calories)		<30	
Saturated fat (% of calories)		<10	
Cholesterol (mg/d)		<200	
Carbohydrate (% of calories)		50-60	
Protein (g/kg/d, % of calories)			
No diabetes	1.4 (~18)		0.6-0.8 (~8-10)
Diabetes	0.8 (~10)		0.6-0.8 (~8-10)
Phosphorus (g/d)	1.7		0.8-1.0
Potassium (g/d)	>4		2.4

Note: Adapted from the DASH diet and NKF-KDOQI™ CPGs for Hypertension and Antihypertensive Agents in CKD, modified for diabetes and stages of CKD.[5,199]

*Adjust so total calories from protein, fat, and carbohydrate are 100%. Emphasize such whole-food sources as fresh vegetables, whole grains, nuts, legumes, low-fat or nonfat dairy products, canola oil, olive oil, cold-water fish, and poultry.

Ø Nutritional recommendations patients with CKD + HBP:(134)

- ü Dietary and other therapeutic lifestyle modifications are recommended as part of a comprehensive strategy to lower blood pressure and reduce CVD risk in CKD.
- ü In fact, individuals with CKD have significant comorbid conditions in addition to high blood pressure, for which dietary modifications are recommended, including diabetes, CVD, obesity, and hyperlipidemia.
- ü Therefore, NKF Guidelines, compiles dietary modifications recommended for the general population as they apply to CKD Stages for management of high blood pressure (table 18).
- ü Dietary sodium restriction is recommended to reduce ECF volume expansion and lower blood pressure. Based on the results of the studies DASH and DASH-Sodium Trials, the Work Group recommended that most individuals with CKD should reduce sodium intake to less than 100 mmol/d (2.4 g/d). Further reduction in sodium intake to <50 mmol/d (<1.2 g/d) might lower blood pressure further, but may be more difficult to achieve.

Table 21: Recommended mineral and micronutrient intakes in CKD patients with hypertension

Nutrient	Stage of CKD	
	Stages 1-4	
Sodium (g/d)*	<2.4	
Total Fat (% of calories)	<30	
Saturated Fat (% of calories)	<10	
Cholesterol (mg/d)	<200	
Carbohydrate (% of calories)**	50-60	
	Stages 1-2	Stages 3-4
Protein (g/kg/d, % of calories)	1.4 (~18)	0.6-0.8 (~10)
Phosphorus (g/d)	1.7	0.8-1.0
Potassium (g/d)	>4	2-4

*Not recommended for patients with "salt-wasting."
**Adjust so total calories from protein, fat and carbohydrate is 100%.

Ø Nutritional recommendations in children :

ü Regular evaluation of nutritional status and Provision of adequate nutrition are key components in the overall management of children with CKD. Thus, the focus of nutritional care for children across the spectrum of CKD must always be centered on the achievement of the following goals:

- Maintenance of an optimal nutritional status to achieve a normal pattern of growth and body composition by intake of appropriate amounts and types of nutrients.
- Preventing the development of Poor-Energy malnutrition (PEM) and meet the children's vitamin and Mineral needs.
- Avoidance of uremic toxicity and metabolic abnormalities.
- Reduction of the risk of chronic morbidities and mortality, later in adulthood.

ü In children with chronic kidney disease (CKD), there is no one-size-fits-all diet that can be prescribed. Medical nutrition therapy needs to be individualized and adjusted as the needs change in a growing child, progression of CKD, and initiation and modality of dialysis. Considerations include calories, protein, sodium, potassium, calcium, phosphorus, and iron:

ü Energy needs: [135]

- Poor energy intake is common in children With CKD stages 2 to 5, due to reduced appetite and vomiting, in fact, Studies have shown that the caloric intake of infants and young children with CKD is frequently less than 80% of recommended intake.[136-138] This fact has been considered to be incriminated in children's growth retardation and development disorders.[139] In addition, these low intakes and Decreased rates of weight gain and growth, may Occur early in those with CKD and worsen with Increasing severity of

CKD.[136][140][141]Thus, the energy requirements for children with CKD stages 2 to 5, should be as high as possible and be adjusted individually according to the height age, body mass and size and not only to the chronological age, further the adjustment based upon the response in rate of weight gain or loss.

- However, there's no evidence that energy requirements of children with CKD are higher than those of healthy children. Likewise, an increase of caloric intake over 80% of recommendations led to no additional effect on CKD children growth.[142]
- A balance of calories from carbohydrate (45 to 50%) and unsaturated fats (40 to 45%) within the physiological ranges, are the dietary recommended intakes suggested when prescribing oral, enteral, or parenteral energy supplementation to children with CKD stages 2 to 5
- Supplemental nutritional support should be considered when the usual intake of a Child with CKD stages 2 to 5 or 5D fails to meet his energy requirements and/or the child is not achieving expected rates of weight gain and/or growth for age.
- A smaller percentage of children have Excessive energy intake, in that case, dietary intervention and lifestyle changes are needed to control the overweight or obesity and to address the short- and long-term of their complications.
- The following tables show the estimated dietary energy intakes advisable by The American National Kidney and Urologic Diseases Information Clearinghouse (NKUDIC), and those recommended by NKF-K/DOQI Guidelines.

Table 22: estimated energy intakes in CKD children according to the (NKUDIC)[143]

Energy Needs for Children with Kidney Disease			
Age Range		Calories / Pound / Day	
Infant	0–6 months	49	
	7–12 months	45	
Toddler	1–3 years	46	
Child	4–6 years	41	
	7–10 years	32	
Adolescents		Girls	Boys
	11–14 years	21	25
	15–18 years	18	20

Table 23: estimated energy intakes in CKD children according to the NKF-K/DOQI[144]

	Age (y)	kcal/kg/d
Infants	0-0.5	108
	0.5-1	98
Children	1-3	102
	4-6	90
	7-10	70
Males	11-14	55
	15-18	45
	18-21	40*
Females	11-14	47
	15-18	40
	18-21	38*

*Based on Recommended Dietary Allowances and increased physical activity.

ü Protein needs: (135)

- The recommendation for protein intake in children with CKD has to consider maintenance of Growth and an adequate nutrition status, but it has also to take in consideration the following risks :

- The intrinsic link of protein intake and phosphorus load. An excessive protein intake is accompanied within an increase of the phosphorus load, resulting in hyperparathyroidism and osteodystrophy.
- Increasing dietary protein intake, means a rise of acid load and then acidosis development, real CKD threat, especially that it can worsen the CKD children's growth disorders.
- High protein intake induces also, an increase in glomerular filtration, which can lead to renal sclerosis and thus worsening even more the kidney disease.

- It is suggested to maintain dietary protein intake at 100% to 140% of the DRI for ideal body weight in children with CKD stage 3 and at 100% to 120% of the DRI in children with CKD stages 4 to 5.

- In children with CKD stage 5D, it is Suggested to maintain dietary protein intake at 100% of the DRI for ideal body weight plus an allowance for dialytic protein and amino acid losses.
- The following table details the different estimated DPI for each age range in children with CKD.

Table 24: estimated protein intakes in CKD children (145)

Protein Needs for Children with Kidney Disease					
Age Range		Grams / Pound / Day			
		Pre-Dialysis	Hemo-dialysis		Peritoneal Dialysis
Infant	0–6 months	1	1.2		1.3–1.4
	7–12 months	0.73	1.1		1.0–1.1
Toddler	1–3 years	0.5	0.7		0.9
Child	4–6 years	0.5	0.7		0.9
	7–10 years	0.45	0.6		0.8
Adolescents	11–14 years	0.45	0.6		0.8
	15–18 years	0.4	Girls	Boys	0.6–0.7
			0.5	0.6	

ü Sodium, Fluid& potassium intakes:(135)(145)

- Fluid and electrolyte requirements of individual children vary according to their primary Kidney disease, degree of residual kidney function, and method of kidney replacement therapy. Supplementation or restriction of fluid, sodium, and potassium intake is individualized and influenced by the volume of urine output and the ability to concentrate urine, hydration status, and the presence or absence of hypertension or hyperkalemia. Dietary and other therapeutic life style modifications are recommended as part of a comprehensive strategy to lower blood pressure and reduce CVD risk in those with CKD.(146)

- Since the most common causes of CKD in children are associated with excessive loss of sodium and chloride, the restriction of sodium and/or fluid seems to be not always appropriate. In fact some infants and children with obstructive Uropathy or renal dysplasia have polyuria, polydypsia, and difficulties to conserve sodium and chloride. These children develop a salt-wasting state and require then salt supplementation. Hence, supplemental free water and sodium supplements should be considered for children with CKD stages 2 to 5 and polyuria to avoid chronic intravascular depletion and to promote optimal growth.
- Individualized therapy can be started by first prescribing at least the age-related DRI of sodium and chloride (table 25), then restriction of sodium intake will be considered for children with CKD stages 2 to 5 and 5D who have hypertension (systolic and/or diastolic blood pressure $\geq$ 95th percentile) or prehypertension (systolic and/or diastolic blood pressure $\geq$ 90th percentile and < 95th percentile). The restricted sodium intakes value will be adjusted according to the blood biochemistry tests results and blood pressure.
- Fluid intake should be restricted in children with CKD stages 3 to 5 and 5D who are oligoanuric to prevent the complications of fluid overload.
- Potassium intake should be limited for children with CKD stages 2 to 5 and 5D who have or are at risk of hyperkalemia.

Table 25 DRI for Healthy Children for Water, Sodium, Chloride and Potassium(145)

	Total Water* (L/d)		Sodium† (mg/d)		Chloride (mg/d)		Potassium (mg/d)	
Age	AI	Upper Limit	AI	Upper Limit	AI	Upper Limit	AI	Upper Limit
0-6 mo	0.7	ND	120	ND	180	ND	400	ND
7-12 mo	0.8	ND	370	ND	570	ND	700	ND
1-3 y	1.3	ND	1,000	1,500	1,500	2,300	3,000	ND
4-8 y	1.7	ND	1,200	1,900	1,900	2,900	3,800	ND
9-13 y	2.4	ND	1,500	2,200	2,300	3,400	4,500	ND
14-18 y	3.3	ND	1,500	2,300	2,300	3,600	4,700	ND

Abbreviation: ND, not determined.

Source: Health Canada: http://www.hc-sc.gc.ca/fn-an/alt_formats/hpfb-dgpsa/pdf/nutrition/dri_tables-eng.pdf. Reproduced with the permission of the Minister of Public Works and Government Services Canada, 2008.

*Total water includes drinking water, water in beverages, and water that is part of food.

†Grams of sodium × 2.53 = grams of salt; 1 teaspoon salt = 2,300 mg sodium.

ü Iron intakes:

- Dietary iron intake is almost always not fully-enough to meet children's needs, because of the occult blood loss in the gastrointestinal tract and possibly the restriction of meat consumption. An oral and/or enteral iron supplements is therefore necessary, particularly in combination with a treatment by erythropoietin.

ü Bone minerals and Vitamin D requirements: (135)

Calcium:

- The management of oral and/or enteral calcium intake in children with CKD is a challenging problem for physicians and dietitians, because both insufficient and excessive calcium supply may occur, and lead either to deficient mineralization of the skeleton in case of insufficient calcium supply, or to severe vascular morbidity in case of calcium overload.
- Therefore, In order to prevent osteodystrophy, the K/DOQI guidelines recommend that In children with CKD stages 2 to 5, the total calcium intake from nutritional sources and phosphate binders must be in the range of 100%

to 200% of the DRI for Calcium for age. This DRI varies from 420 to 2500mg per day, in accordance with the age.

- The use of a calcium supplement of 0.5 to 1g/m^2/24h to augment inadequate oral and/or enteral calcium intake should be considered when children with CKD stages 2 to 5 and are unable to meet their protein requirements through food and fluids alone.(147)
- See Table 26 for dietary calcium intakes, estimated for CKD children.

Viatmin D:

- Recent clinical evidence suggests a high prevalence of vitamin D insufficiency in children and adults with CKD, thereby, it is recommended that InchildrenwithCKDstages2to5, serum25-hydroxyvitaminDlevels be measured Once per year. If the serum level of 25-hydroxy vitamin D is less than 30ng/mL (75 nmol/L), supplementation with vitamin D2 (ergo calciferol) or vitamin D3 (cholecalciferol) is suggested.
- In the repletion phase, it is suggested that serum levels of corrected total calcium and phosphorus be measured at 1 month following initiation or change in dose of vitamin D and at least every 3 months thereafter.
- When patients are replete with vitamin D, it is suggested to supplement vitamin D continuously and to monitor serum levels of 25-hydroxyvitamin D yearly.
- See Table 28 for recommended supplements for Vitamin D deficiency in children with CKD.

Phosphorus:

- The NKF-K/DOQI guidelines concerning phosphorus intakes are as follows:
- In children with CKD stages 3 to 5, reducing dietary phosphorus intake to 100% of the DRI for age is suggested when the serum PTH concentration is above the target range for CKD stage and the serum phosphorus concentration is within the normal Reference range for age.
- In children with CKD stages 3 to 5 and, reducing dietary phosphorus intake to 80% of the DRI for age is suggested when the serum PTH concentration is above the target range for CKD stage and the serum phosphorus concentration exceeds the normal reference range for age.
- After initiation of dietary phosphorus restriction, it is suggested that serum phosphorus concentration be monitored at least every 3 months in children with CKD stages 3 to 4 and monthly in Children with CKD stage 5, in all CKD stages, it is suggested to avoid serum phosphorus concentrations both above and below the normal reference range for age.
- See Table 27 for estimated daily intakes of phosphorus, in children with CKD.

Table 26 Recommended Calcium Intake for Children with CKD Stages 2 to 5 and 5D

Age	DRI	Upper Limit (for healthy children)	Upper Limit for CKD Stages 2-5, 5D (Dietary + Phosphate Binders*)
0-6 mo	210	ND	≤420
7-12 mo	270	ND	≤540
1-3 y	500	2,500	≤1,000
4-8 y	800	2,500	≤1,600
9-18 y	1,300	2,500	≤2,500

Abbreviation: ND, not determined.
*Determined as 200% of the DRI, to a maximum of 2,500 mg elemental calcium.

Table 27 Recommended Maximum Oral and/or Enteral Phosphorus Intake for Children With CKD

		Recommended Phosphorus Intake (mg/d)	
Age	DRI (mg/d)	High PTH and Normal Phosphorus*	High PTH and High Phosphorus†
0-6 mo	100	≤100	≤80
7-12 mo	275	≤275	≤220
1-3 y	460	≤460	≤370
4-8 y	500	≤500	≤400
9-18 y	1,250	≤1,250	≤1,000

Source: Health Canada: http://www.hc-sc.gc.ca/fn-an/alt_formats/hpfb-dgpsa/pdf/nutrition/dri_tables-eng.pdf. Reproduced with the permission of the Minister of Public Works and Government Services Canada, 2008.
*≤ 100% of the DRI.
†≤ 80% of the DRI.

Table 28 Recommended Supplementation for Vitamin D Deficiency/Insufficiency in Children with CKD

Serum 25(OH)D (ng/mL)	Definition	Ergocalciferol (Vitamin D_2) or Cholecalciferol (Vitamin D_3) Dosing	Duration (mo)
<5	Severe vitamin D deficiency	8,000 IU/d orally or enterally × 4 wk or (50,000 IU/wk × 4 wk); then 4,000 IU/d or (50,000 IU twice per mo for 2 mo) × 2 mo	3
5-15	Mild vitamin D deficiency	4,000 IU/d orally or enterally × 12 wk or (50,000 IU every other wk, for 12 wk)	3
16-30	Vitamin D insufficiency	2,000 IU daily or (50,000 IU every 4 wk)	3

Note: Conversion factor for Serum 25(OH)D: ng/mL × 2.496 = nmol/L.
Adapted with permission.[121]

Ø Nutritional management of kidney transplant patients :(148)

- After kidney transplant, patients should continue to require dietary modifications, in order to address nutrition related issues, prevent post-transplant complications and particularly to prevent the side effects of immunosuppressive medications, that significantly impact on the long-term morbidity and life quality of kidney transplant recipients.
- In adult patients with renal transplant, dietary management has the same objectives as in moderate chronic renal failure(149), while transplant children require a dietary management of protein and phosphorus in the same way as children with similar GFR before transplantation.(135)
- Dietary protein requirements: (148)

- Guideline objective: To ensure that appropriate nutrition interventions are used to prevent the loss of lean body mass and achieve neutral or positive nitrogen balance in kidney transplant recipients, and to reduce the risk of chronic kidney failure in stable kidney transplant recipients.

- Practice recommendations:

1. In the first four weeks after transplantation, kidney transplant recipients should be advised to eat a diet providing 1.3g protein/kg body weight to reverse negative nitrogen balance and maintain lean body mass.
2. Kidney transplant recipients with chronic rejection should restrict dietary protein to 0.73+/-0.11g/kg body weight.
3. Stable kidney transplant recipients on a maintenance immunosuppression regimen, irrespective of renal function, should not exceed the recommended daily intake of protein for general population of 0.75g protein/kg body weight for females and 0.84g protein/kg body weight for males.
4. During periods when treatment with high dose prednisone is required, for example during acute rejection, protein requirements may be elevated to a level similar to that of the early post-transplant period.
5. Regular review by a dietitian is desirable in the long term to ensure that protein requirements are neither exceeded (particularly in the presence of chronic nephropathy) nor inadequate (particularly in periods of acute rejection when prednisone dose may be increased).

- Evidences:

1. The study by Cogan et al[150] which examinated nitrogen balance in kidney transplant recipients requiring dialysis immediately after transplant, confirms that prednisone increases protein requirements. To correct negative nitrogen balance, patients required 1.3+/-0.06g protein/kg/day.
2. the recommendation that 1.4g protein/kg body weight be consumed in the early post-translpant period is based on single study by Whittier et al[151], in which patients achieved positive nitrogen balance, when they consumed at least 1.4g protein/kg/day. However, it should be noted that patients in this

study received 1mg prednisone/kg/day for the first two weeks post-transplant, followed by a progressive reduction of prednisone to 0.5 to 0.7mg/kg/day over the second two-weeks period.

- Dietary Sodium requirements:[148]

- Guideline objective: To ensure prevent and manage hypertension in adult kidney transplant recipients.

- Practice recommendations:

1. Hypertensive kidney transplant recipients should be advised to restrict sodium intake to 80-100mmol/day.
2. Lowering sodium intake to 65-70mmol per day may cause a greater lowering of blood pressure.
3. kidney transplant recipients should be encouraged to do at least 30 minutes of moderate intensity physical activity on at least five days per week.

- Evidences:

1. The recommendation to limit sodium to 80-100mmol/day is in the line with current guidelines for the general population.[152] However, clinicians should emphasise adequate fluid intake over sodium restriction in the immediate post-transplant period.
2. The recommendation to lower sodium intake further to 65-70mmol/day is in the line with suggested dietary target for chronic disease prevention set by the National Health and medical research Council and the New Zealand Ministry for health, and recently adopted by the national Heart foundation of Australia.[153]
3. the practice recommendations above are in the line with guidelines for hypertension management in the general population. In the general population, regular aerobic activity and weight reduction by as little as 5kg

reduces blood pressure in most people who are greater than 10% above their ideal body weight. (154)

- Dietary Calcium & Vitamin D requirements: (148)

- Guideline objective: To minimize bone mineral density loss and reduce the risk of fractures among adult kidney transplant recipients.

- Practice recommendations:

1. Kidney transplant recipients should be advised to take a Vitamin D supplement at a dose of at least 0.25ug daily.
2. A diet containing adequate calcium-rich foods to meet the recommended dietary intake for calcium of 1000mg/day (1300mg/day post-menopause), should be encouraged. If the diet does not provide adequate calcium, a calcium supplement should be recommended.

- evidences:

1. The meta-analysis of the all available trials (24 trials, 1299 patients) combined, shows that any intervention (Vitamin D sterol, or Bisphosphonates, or...) for bone disease in kidney transplant recipients, does reduce the risk of fracture and provide also a significant improvement in bone mineral density, in this population.(155
2. Two randomized controlled trials have shown that bisphosphonates combined with a calcium intake of 1000mg/day, have greater efficacy to preserve bone mineral density at the lumbar spine and femoral neck than Vitamin D sterols associated with calcium.(156)

- Dietary Phosphorus requirements:(148)

- Guideline objective: To correct hypophosphataemia in adult kidney transplant recipients.

- Practice recommendations:

1. Supplementation should commence as soon as possible after transplantation to correct hypophosphoataemia.
2. The treating physicians should determine the dose on the basis of serum phosphate concentrations. Otherwise, If serum phosphate level is normal, the recommended phosphorus intake should be no less than 1.2g per day.(149)
3. Physicians should be aware that in the late post-transplant period phosphate supplementation has the potential to worsen hyperparathyroidism and may mask phosphorus deficiency.

- Evidences:

There have been no studies examining the effect of dietary phosphate intake on hypophosphataemia in kidney transplant recipients. The general consensus among experts, is that the intake of phosphate-rich foods should be encouraged as early as possible.

- Dietary guidelines summary:[148][149]

Nutrient daily intakes in kidney transplant recipients:

- First month after kidney transplantation :

  Proteins: 1.3g/kg/day

  Calories: 30-35 Kcal/day

- In chronic rejection:

  Proteins: 0.73+/-0.11g/kg body weight.

- In stable kidney transplant:

- Proteins: 0.75g/kg/day
- Sodium: 80-100mmol/day
- Calcium: 1000mg
- Vitamin D: 0.25ug/day
- Phosphorus: 1.2g/day
- Iron: 300mg/day
- Carbohydrates 50% of calories
- Fat 30% of calories
- 300mg cholesterol /day
- Report polyunsaturated fatty polyacids / saturated> 1g

9.4 Malnutrition in CKD:

9.4.1 Definition:

- Malnutrition in the context of CKD, is a condition that develops when the body get an inappropriate dietary intake of one or more macro- or micro-nutrients -necessary to maintain healthy tissues and organ function- and which results in metabolic abnormalities that cannot be corrected solely by an improved diet. (157)
- Thus, malnutrition may indicate both a state of under-nutrition or over-nutrition. Under-nutrition is “a consequence of consuming too few essential nutrients or using or excreting them more rapidly than they can be replaced” ; whereas over-nutrition is usually a result of “eating too much, eating too many of the wrong things, not exercising enough or taking too many vitamins or other dietary replacements”.(157)
- In patients with ESKD, uraemic malnutrition generally refers to patients with kidney disease, who have suboptimal nutritional status characterized by reduced body weight, depleted fat stores, loss of somatic protein (muscle mass) and low levels of visceral proteins such as s-albumin, pre-albumin and transferrin. (158)

9.4.2 Causes & mechanism of malnutrition in CKD:

Ø Mechanism:

- Nutritional and metabolic derangements are common in CKD and play a major role in affecting clinical outcome in this patient population.(159) The development of impaired nutritional status, in Stage 4 and 5 CKD, commonly termed uraemic malnutrition, is multi-factorial. The term uraemic malnutrition is used to encapsulate the dietary and metabolic changes which lead to a state of protein catabolism and loss of lean body mass. Malnutrition is typically

hallmarked by a progressive decline in body weight and body protein.(160) It is the loss of metabolically active component of body mass which creates the negative effects seen with malnutrition.(161)(162)

- Figure 23(163) provides a schematic diagram highlighting the complex interrelationships of factors leading to uraemic malnutrition in CKD. Table 29(164) provides an overview of the metabolic consequences of a number of conditions which may contribute to the development of uraemic malnutrition inCKD.

Table 29 Mechanisms for the progression of uraemic malnutrition in CKD

Cause	Mechanism
Reduced dietary intake	Poor appetite from uraemic toxicity, inflammation, reduction in anabolic hormones[17]. Spontaneous decline in energy and protein intake[18]
Restrictive dietary intake	Unmonitored restriction of protein and other nutrients
Inflammation	From whole-body protein catabolism, suppressed albumin synthesis, anorexia, disrupted growth hormone and IGF-1 (decreased anabolism)[3], increased resting energy expenditure[19].
Gastrointestinal function	Delayed gastric emptying, impaired motility, alterations in bacterial flora[20]
Metabolic acidosis	From stimulation of ubiquitine-mediated proteolysis, resulting in a negative nitrogen balance, catabolism of branched-chain amino-acids, muscle protein and suppress albumin synthesis[21]
Abnormal hormone response	Altered carbohydrate and lipid metabolism, insulin resistance and metabolic acidosis[22]

Ø causes:

Renal causes of malnutrition:

The kidneys are central to various metabolic processes, including the recycling of amino-acids from small circulating peptides, the re-uptake of glucose and the metabolism of waste products from the urea cycle. As renal function decreases, the accumulation of waste compounds occurs, resulting in a toxic concentration of potentially harmful substances. This state of uremic intoxication, also known as uremia, is both detrimental to systemic homeostasis and has detrimental consequences for more complex metabolic processes such as appetite regulation and food intake.(165)(166)

Chronic low-grade inflammation:

Much research demonstrate chronic low-grade inflammation as a causal factor of malnutrition in CKD(167). It has been reported that inflammation is present in 30 to 50% of ESRD patients;(168) furthermore, inflammation is an important cause of hypoalbuminaemia,(169) which is a risk factor for mortality in these patients.(170)(171) Inflammation has been shown to decrease protein production and to increase protein breakdown, resulting in a highly negative protein balance.(172) Indeed, Stenvinkel et al. found markedly higher prevalence of inflammation in patients with cardiovascular disease (CVD) and malnutrition terming this commonly observed triad of concurrent complications as malnutrition, inflammation and arteriosclerosis (MIA) syndrome,(168)(173) and others have made similar observations.(174)

Metabolic acidosis:

Metabolic acidosis is a common metabolic disturbance in CKD patients that predispose to bone disease, altered protein metabolism, skeletal muscle wasting, and progressive glomerular filtration rate loss.(175) In addition, acidosis

induces increased protein catabolism with degradation of the essential, branched-chain amino acids and muscle protein.(176) Moreover, metabolic acidosis is also known to suppress synthesis of proteins such as albumin,(176) and low levels of albumin as well as catabolism are strong predictors of mortality. Indeed, metabolic acidosis, generally manifested by reduced serum bicarbonate levels, is also correlated with increased mortality.(177)(178) Interestingly, Kovesdy et al. found that not only low levels of bicarbonate (<22 mmol/L) are associated with mortality, but also high levels (>29 mmol/L)(179).

Dialysis treatment:

Dialysis treatment has undoubtedly prolonged CKD patients´ lifespan and improved uremic intoxication commonly found in pre-dialysis patients. However, these benefits come with potential side-effects. A detailed review of the literature(180) have shown that PD therapy induces a decreased turnover rates and a reduced efficiency of protein turnover. This condition is especially harmful under stress as nutrient intake may be insufficient especially during superimposed catabolic illnesses.(180) In HD patients, many factors that influence protein metabolism predispose these patients to increased catabolism and loss of lean body mass. It has been shown that the HD procedure in itself can induce protein catabolism, these adverse effects are likely the result of decreased protein production and increased protein breakdown.(181)(182) Indeed, low plasma amino acid levels during HD sessions are thought to be one of the possible explanations for increased catabolism.(182)

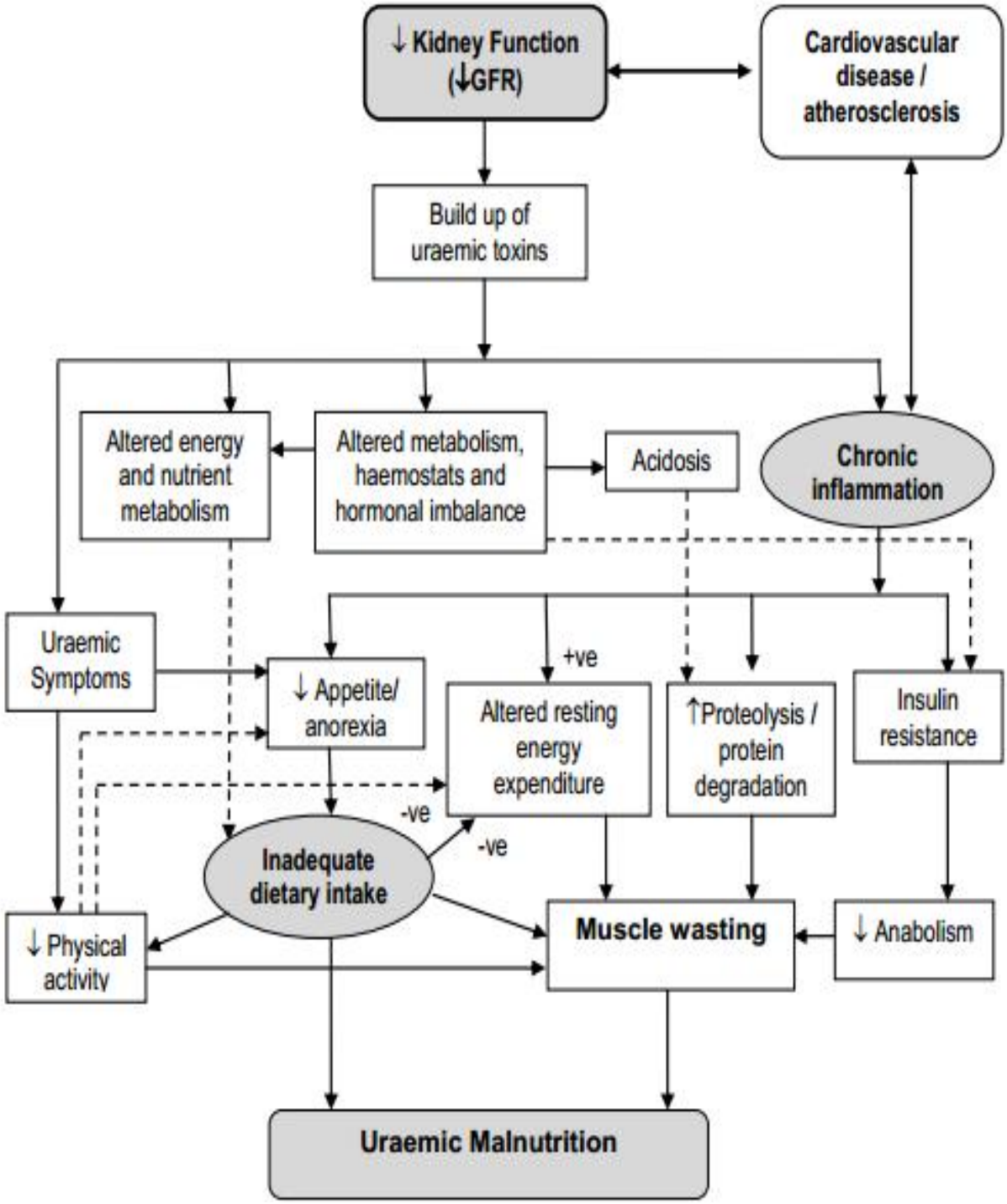


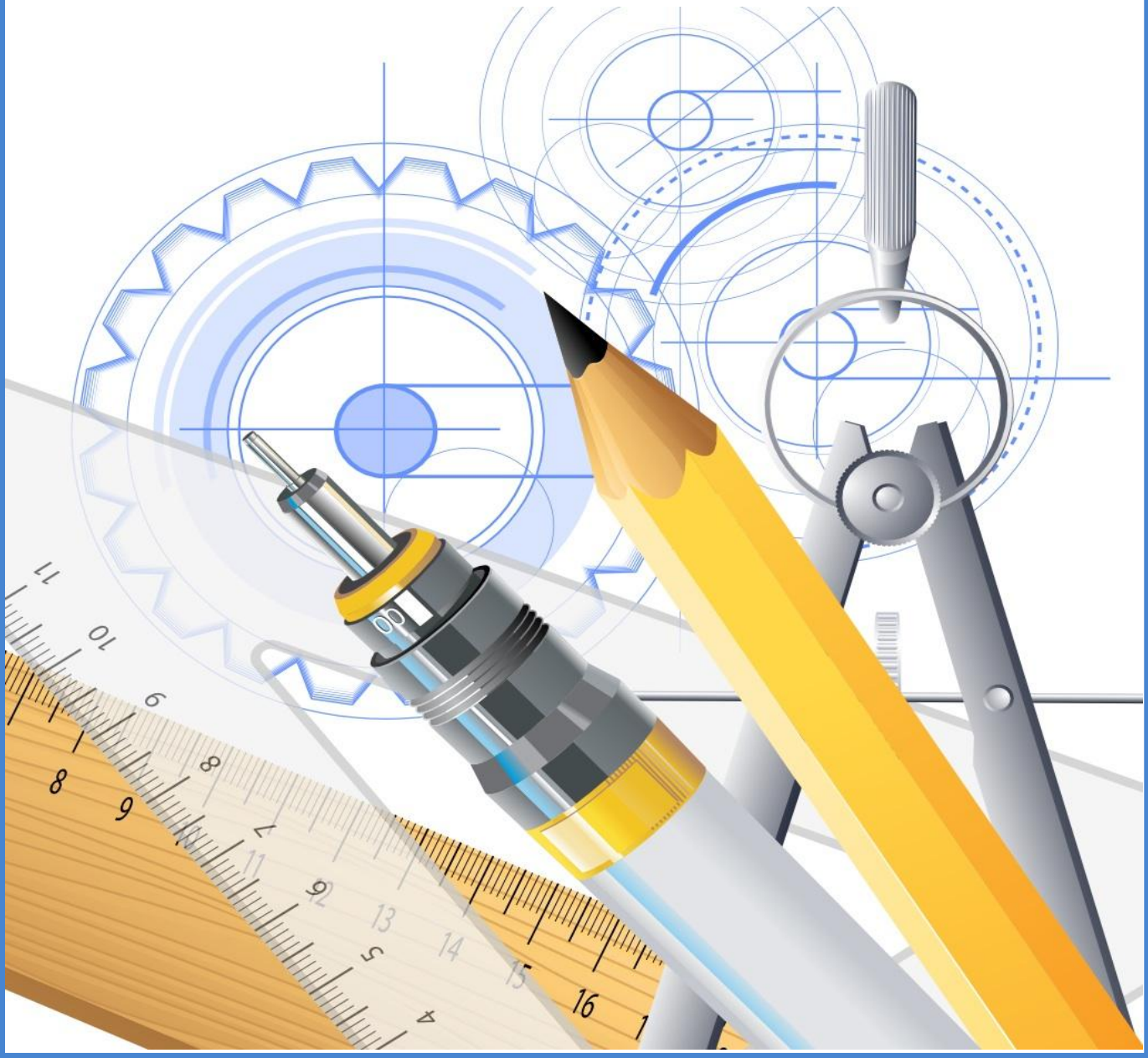
Figure 23: Mechanism for the development of uraemic malnutrition in chronic kidney disease

9.4.3 Consequences of malnutrition in CKD:

Malnutrition in dialysis is associated with high mortality.(183) Indeed, the annual mortality of hemodialysis malnourished patients is around 30% while it is generally 10%. Note that in this population, overweight and obesity paradoxically appear as a good prognostic factor.(184)

Finally, malnutrition can also further exacerbate declining renal function. Firstly, due to a reduction in the renal blood (plasma) flow, GFR and urine concentrating capacity of the residual function is further reduced as a consequence of malnutrition. In addition, acid/base regulation and sodium balance may be further impaired.(185)

MATERIAL & METHODS



Tools & methods to create the application

The design& development of a Moroccan self-management application, for nutrition in chronic kidney failure, have required many primary measures before the realization step, notably building a work team, dividing tasks and evaluating the feasibility of the project.

1. The work team selection:

- ü The work team was formed during the 1st International Workshop on Engineering and Innovation in Healthcare (WEITH'15), that took place on 2d December 2015, in the faculty of sciences & technologies of Fez (FST).
- ü By the end of the forum, the team was thereby, made of the following members:

- Professor and headmaster of nephrology department in the university hospital Hassan 2 of fez.
- I.T engineer and professor in the faculty of sciences &technologies of Fez.
- The dietitian affected in nephrology department of Hassan 2 hospital.
- Two engineering students in the FST of Fez.
- And a medical student in the graduation phase, from the faculty of medicine and pharmacy of Fez.

- ü Several concertation meetings of the team, have been organized at the nephrology department of Hassan 2 hospital, in order to set up a demarche for the realization of the project. In fact, it was decided that the Smartphone application will be developed according to the engineering approach described by Denning(186), which follows the processes mentioned below:

- Design process
- Development /Encoding process
- Implementation/experimentation process
- Evaluation/Validation process

ü The headmaster of nephrology department with the dietitian and the medical student, were responsible for the application content & design, while the engineering professor within the engineering students, formed the subgroup in charge of encoding part of the project.

2. Design process:

2.1 Tools & methods for database collection:

ü After numerous reunions of the team in charge of the application design, and which took place over a period of 3 months, the preliminary content of the application was developed based on:

- The National Kidney Foundation (NKF) latest recommendations about GFR estimation formulas, in addition to the latest published articles regarding renal function assessment (61, 62, 63,64,65,66,67,68,69, 187,188,189).
- The latest nutritional guidelines advised by (122 -> 156) :

- National Kidney Foundation, Disease Outcomes Quality Initiative (DOQI).
- National Agency for accreditation and healthcare evaluation (ANAES).
- European Society for Parenteral and Enteral Nutrition (ESPEN).
- European Best Practice Guidelines (EBPG).
- European Dialysis and Transplant Nurses Association/European Renal Care Association EDTNA/ERCA.

- General Repertory of Food (Repertoire general des aliments) 2d edition (editors INRA editions), CNVA-CIQUAL (Lavoisier TEC/DOC), Table CIQUAL 2008 Website AFSSA.
- The international dietary guide book for patients with kidney failure (For recipes).
- The Design & content of nutrition mobile applications, developed in France (Nutridial application by Sanofi france), and United States (KidneyAppetite application by Sanofi renal U.S). (190,191)
- Interactive website for food survey. (192)

2.2 Tools & methods for the application design:

ü To carry up the design of the application, below the specific design tools were used in this thesis project:

- *ArgoUML*: Design software to creat diagrams UML, that itemizes the user's path on the application.
- *Enterprise Architect* : Modeling and design software, to create the varied interfaces of the application.
- *Adobe Photoshop CC 2014*, and *Microsoft paint 2015* : To create all the pictures and frames essential for the application interfaces.

3. Development process:

ü The development of the application was insured by the two engineering students and supervized by the I.T professor, from the faculty of sciences & technologies of Fez.Many other gatherings have allowed to harmonize the

language and especially to study the technical feasibility and define the rules &criterias that should be respected all along the application encoding:

- Accessibility: The user access into the app, should be ensured by a simple search and download from Play Store, without constraint of location.
- Usability: the application modeling, should provide clear interfaces with an easy way to navigate on the app and move from a section or menu to an other.
- Security: Confidentiality is a crucial criteria; all the users profiles, accounts and personal informations, must be protected from any external intrusion.
- Personalization: Security rules should not, nontheless, prevent the user's doctor to make any personalized modifications in the "Dietary guidelines" section. Free open space should be left for that matter. Meanwhile, privacy settlement must not deprive the user from sharing his/her confidential data with other users, either on the app, or throw any other social network.

ü The development material used in this thesis project, is *Android Studio.* Which is considered the offcial integrated development environment for Android platform development. (193)

4. Implementation process:

ü As far as the implementation of the application is concerned; assistance, support and accompaniment of all professors, are sought, in order to introduce the application to the rest of the medical staff particularly residents and dietitians, on a hand, and to integrate it among users (Patients) on the other hand. Which will require many training & education sessions?

ü A special program of therapeutic education, for patients with CKD (ETP), has been elaborated in nephrology department of Hassan 2 hospital. The program

was the result of two thesis projects realized on 2012. The workshops take place weekly on Wednesday where patients are taught many themes about renal function, renal diseases, CKD management and nutrition in renal failure with the eventual presence of dietitians. The workshops would be a convenient opportunity for the implementation of the App, to form patients about its several aspects and the way to use it.

- ü So as to facilitate the implementation of the platform even more, a video tutorial will be integrated In the app as a use menu detailing the course of the user on the app.

5. Experimentation &Validation process:

- ü All over a period of 3 to 6 months after implementation; professors, residents and dietitians will be prompted to note periodically, their own remarks and comments regarding any technical problem, thereafter discuss it during staffs or by emailing.
- ü The application developers will be afterwards contacted for eventual modifications.
- ü By the end of validation period, a survey might be conducted among both medical staff and patients, in order to check all the application aspects, either the technical, the practical or the conceptional one.

Tools & methods to assess the need

In order to adapt more, our application to the users' requirements, we needed to study the target population, to assess their needs regarding the application, to figure out the challenges and constraints faced by CKD patients, and to bring out their own suggestions in matter of nutritional self-management in CKD. To achieve those objectives, we opted for a survey methodology, to conduct among CKD patients.

1. Type of the study:

It is a survey that studies a randomized sample among the concerned population, conducted by the mean of a questionnaire as a data collection technique, so as to improve the number and the accuracy of the survey's related responses.

2. Period of the study:

The survey was maintained during 3 successive months; all along the period extending from the 1st July 2016 to the 30th of September 2016.

3. The target population:

The survey has, as a target sample, every patient, adult or children, diagnosed with chronic kidney disease. All CKD stages are included.

3.1 Inclusion criteria:

It is included in this survey, all the adult and children patients with a GFR<60mL/min/1.73m^2 for over three months. Either followed-up, treated, dialyzed or not

3.2 Exclusion criteria:

The only exclusion criteria from the survey, is the disagreement of the patient himself to participate and reply on the questioning.

4. The sample collection:

The sample was collected in a random way, from the following healthcare establishments:

- Nephrology department of the university hospital of Rabat; Avicenne.
- Pediatric department of Rabat's university hospital.
- Hemodialysis Center of Agdal, Rabat.
- Basic healthcare centers located in Meknes city.
- Basic healthcare center of Lmhaya.

5. Number:

We could collect a sample of 100 patients with chronic kidney disease, ranged as follows:

Adults:

- 56 chronically hemodialyzed patients.
- 11 patients with CKD stage 1.

- 6 patients with CKD stage 3.
- 4 patients with CKD stage 2.

Children:

- 9 patients with CKD stage 2.
- 11 patients on maintenance hemodialysis.

6. Data collection:

Throughout a methodical questioning, several parameters were evaluated. The questionnaire consisted of three parts; there are questions about (i) general personal information, (ii) dietary targets controlling and how it alters their life quality and (iii) adherence to medical prescriptions. Questions about basic personal information such as age, gender, education level and CKD stage, were asked in the general personal information part. The second part consisted of questions regarding the control of nutrition intakes; how the subjects manage their daily requirements, whether it is difficult for them or not. The underlying reasons, were also investigated. In the last part, medical compliance and subjects' awareness of its importance were asked. (The questionnaire is included in the Appendix)

- ### Age:

The age is an important parameter to define, since the dietary guidelines vary according to the chronological age. Some factors and conditions are also influenced by the age, such as the patients ability to self-manage their nutritional status, to use the Smartphone and/or to learn about the application, further the desire to ameliorate their life quality, the medical compliance...

- Level of education:

The educational stage reflects the patient's ability to handle his nutritional treatment and how far he/she can manipulate the application. Moreover the faculty of using the English version of the platform.

- CKD stage:

Likewise, the nutrients intakes differ from a stage to an other, whence the necessity of defining the patient's stage of disease.

- The know & the know-how:

The primary information to bring out through the survey, is the patients knowledge about their daily dietary guidelines and the way to reach them. Further their nutritional behavior when they cannot get at their dietary targets (restrictive or excessive behavior), and during the several Moroccan occasions. How all this is affecting their life quality is an other important condition to precise in this investigation.

- Medical compliance:

Examining the patients adherence to their treatment, allows to assess the need for a reminder system for medications.

- Personal suggestions:

Free space was left for patients, to give any useful opinion that could help to meet the need

RESULTS


Recipes
Couscous with veal
Lentils
Rfissa
Pastilla
Chicken
My dietary guidelines
My CKD stage guidelines
Protein 0.6 g/day
Sodium 400-1000 mg/day
Potassium 1500-1600 mg/day
Calcium 1200 mg/day
Phosphorus 600-1000 mg/day
Fluids 1500 ml/day
Calories ≥ 35 kcal/day
More
Recipes
Grocery Items
Fruits
Fish
Grocery Items
Apples
Pears
Oranges
Add
More

1. Design process:

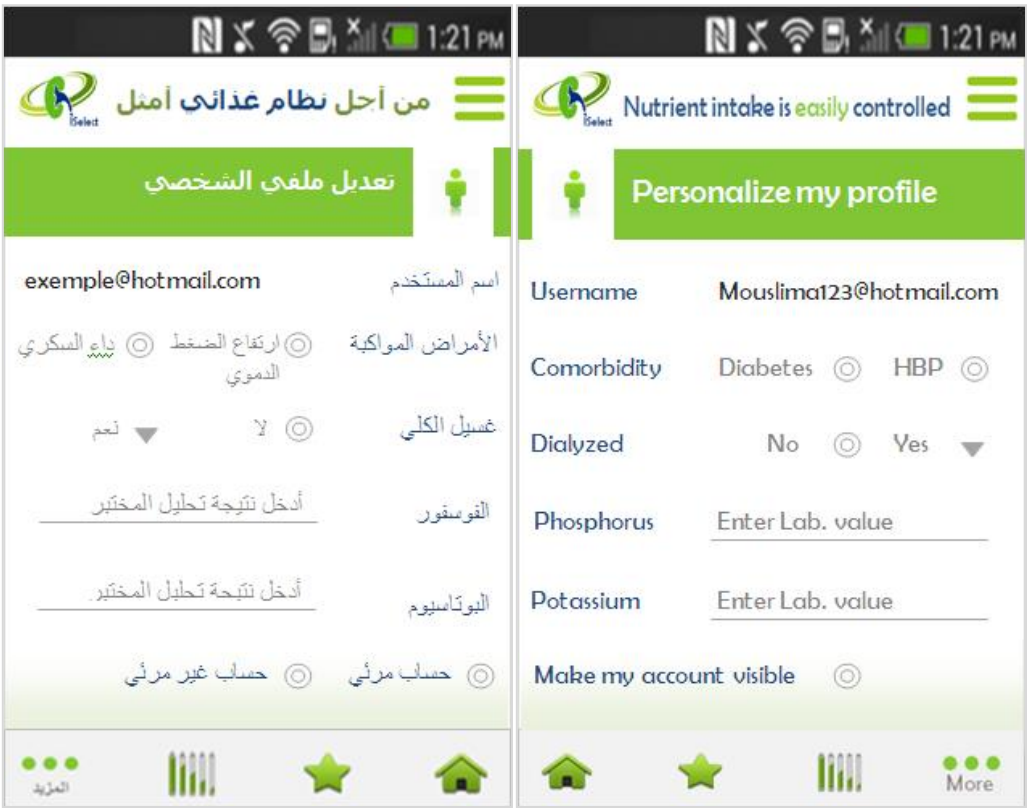
(See the detailed application design in appendices)

- Since the overriding benefit that the platform offers to its users is the “Self” management; the ability to control daily intakes and select food items by them selves, we hence chose *“iSelect”* as a name for the application.
- With the intention of facilitating the user’s navigation on iSelect app, we opted to devide all the options and services provided on the platform, into 3 main menus. Each menu contains many other sections or submenus. Users could easily move from a menu to an other by a simple click on the “Back” or “Home” icons.


1:21 PM
Nutrient intake is easily controlled
Personalize my profile
My eGFR Calculator
My dietary guidelines
Grocery Items
Recipes
My reports
More
Nutrient intake is easily controlled
Medication reminder
Education Tips
Events
Share
Inbox
More
من أجل نظام غذائي أمثل
تعديل ملفي الشخصي
حساب معدل الترشيح الكبيبي
المبادئ التوجيهية الغذائية
خطط وجباتك اليومية
وصفات محلية
تقارير
معلومات عن التطبيق
خروج
المزيد

1.1Personalize my profile:

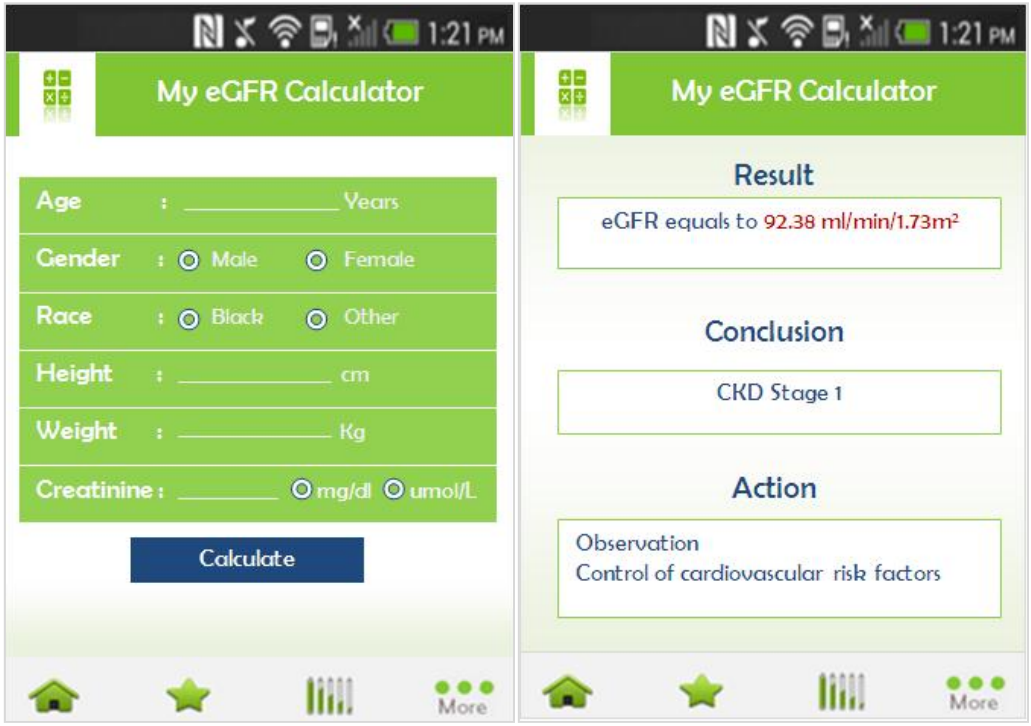
Personalizing each user's account, was an essential need to meet, since all the following options depend on personal data that differs from a patient to an other. The user is supposed to fill in a kind of form, based on his/her medical history, received treatment (on maintenance dialysis or not), and the latest blood analysis results.



Personalize my profile menu (English version) Personalize my profile menu (Arabic version)

1.2 My GFR calculator:

- The first function that iSelect provides, is defining the patient's CKD stage, by calculating the Glomerular filtration rate (GFR). In adult patients, the eGFR will be estimated usingthe three formulas, previously mentioned (Cockcroft-Gault, MDRD and CKD-EPI). Thereafter, the average of the 3 calculated values, will be retained as the real value of GFR. While in children, the estimation of GFR will be based only on *Schwartz formula.*
- The application displays, afterward, the obtained results within the CKD related stage and the varied actions that should be followed in that stage.



My GFR calculator menu (English version)

My GFR calculator menu (Arabic version)

1.3 My dietary guidelines:

This menu allows to every patient with CKD, to be aware of his daily nutritional requirements. A table of dietary guidelines for calories and the six key nutritional values that affect kidney health (Phosphorus – Sodium – Calcium – Protein – Potassium and Fluids), will be displayed in accordance with his/her eGFR previously calculated and his blood tests results, noted on “Personalize my profile” menu.

All dietary guidelines are available, for all CKD stages, for both adults and children, with or without comorbidities (Hypertension, Diabetes).

My dietary guidelines menu (English version)

My dietary guidelines menu (Arabic version)

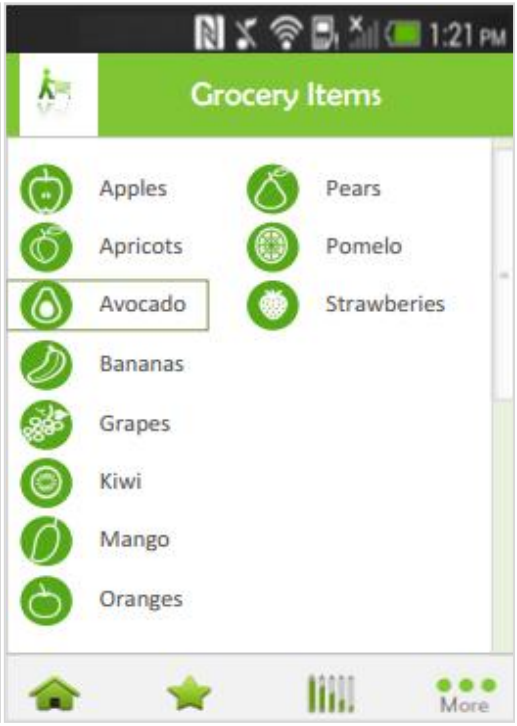
1.4 Grocery items:

Grocery items is an important and rich enough menu, to be considered one of the great services provided on iSelect app; it is actually a huge catalog that integrates all food items classified into 13 principal menus, each menu contains an exhaustive list of foods. A simple click allows to display the varied amounts of nutrients contained in 100mg of each product.

The users could select freely the food items they are about to consume. The entire content of the chosen meal will be calculated and compared with the patient's requirements, in case of it exceeds the recommended guidelines, a warning message will be displayed, yet patients could, however, validate the meal if they want to.

All selected and validated meals will be recorded, so that patients could consult them subsequently, or share them with other patients as well. Patients will have also the option to track and monitor their intakes gradually all along the day.

Grocery items: Mains menus



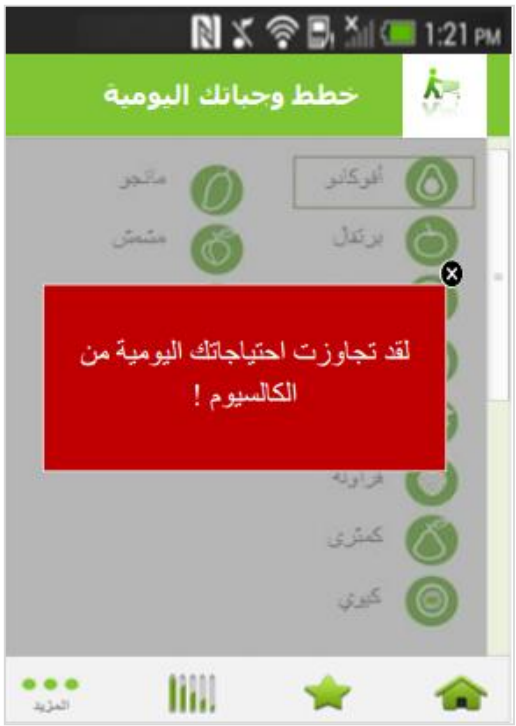
Grocery items: submenus

Grocery items: Composition of the selected food

Follow-up of nutrients consumed during the day

Grocery items: submenus (Arabic version)

Grocery items: Mains menus (Arabic version)



Grocery items: Composition of the selected food (Arabic version)

Follow-up of nutrients consumed during the day (Arabic version)

1.5 Recipes :

Moroccan meals are so diversified and marked by a large range of fruits, vegetables and spices. They contain high amounts of lipids, proteins and minerals, whence the difficulty of limiting dietary intakes in CKD patients. For that matter, iSelect app affords a special catalog of the most famous and frequently consumed, Moroccan recipes. Those recipes come along with micronutrients and mineral contents, and could likewise be added to the daily pannier.

Recipes menu: principal submenu

Recipes menu: recipe

Recipes menu: Composition

Recipes menu: principal submenu (A.V)

Recipes menu: recipe(A.V)

Recipes menu: Composition (A.V)

1.6 My reports :

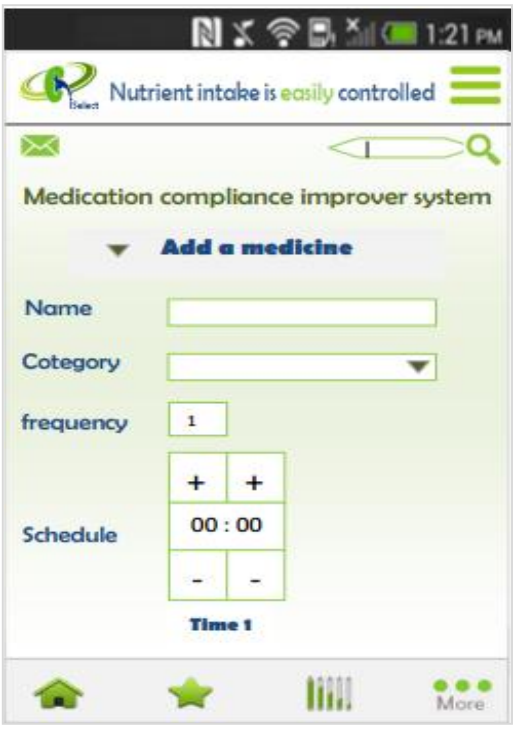
Monitoring the nutritional status of patients with CKD, is evidently one of the leading aims of the application, in fact “My reports” menu has been specifically designed to achieve that objective. It provides special graphics that show how dietary intakes evolve over the past weeks. Thereby, any abnormal facts of over or undernutrition could be detected.

My reports menu (E.V)

My reports menu (A.V)

1.7 MCIS:

On top of self-management services provided on the app, iSelect offers further option in matter of medical observation, in order to enhance polypharmacy related problems. Medical Compliance Improver System(MCIS) is in fact, an electronic sonorous alarm that reminds patients of their medication schedule, that patient should previously fill up in MCIS menu.

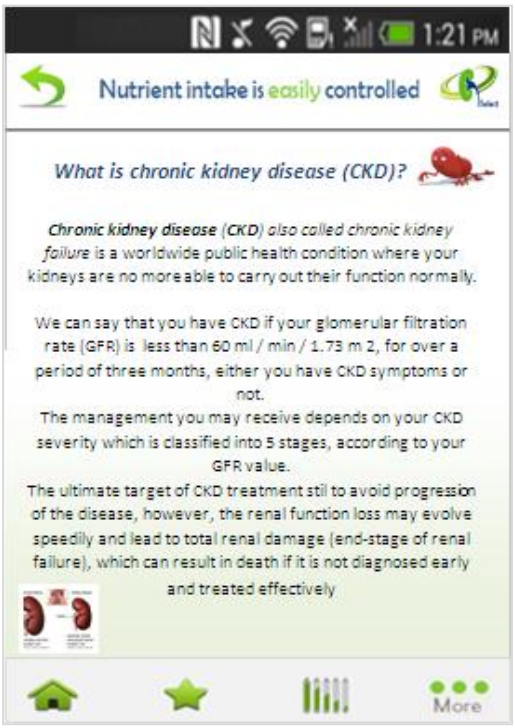


MCIS menu (E.V)

MCIS menu (A.V)

1.8 Education tips:

Education tips menu is a space where users could learn more about their chronic disease, many questions that might interest CKD patients are answered over there, in a simplified way that could easily be apprehended by all cultural levels.



Education tips menu

1.9 Other functions:

- iSelect gives patients the further possibility to share their personal content with other users of the platform by means of mini messages, or with doctors and dietitians by email, or with any other person through social networks.
- During special occasions, patients may compose any particular meal then record it in “Events” menu. All registered meals could be consulted subsequently.

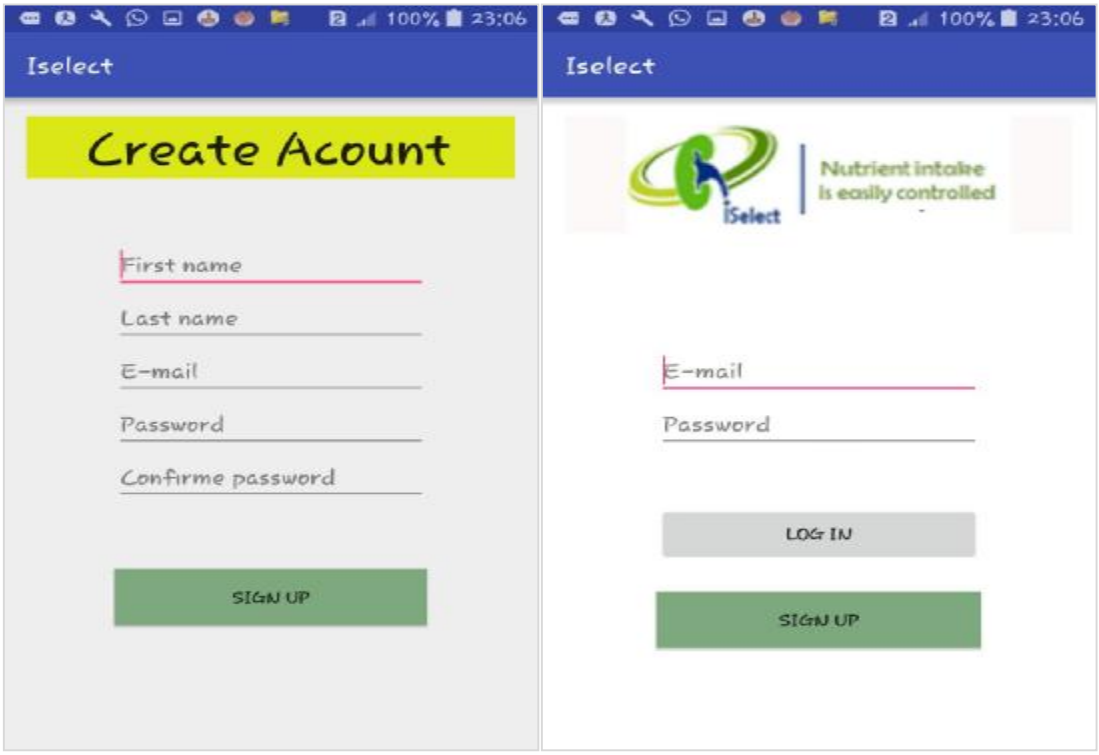
2. Development process:

The majority of English version of iSelect application, have been effectively developed by the work team in charge of the encoding part of the project. The operating system chosen for the platform is Android. All privacy settlements, accessibility and security rules were respected.

The development process gave birth to an easily usable application, with clear interfaces and harmonized content.

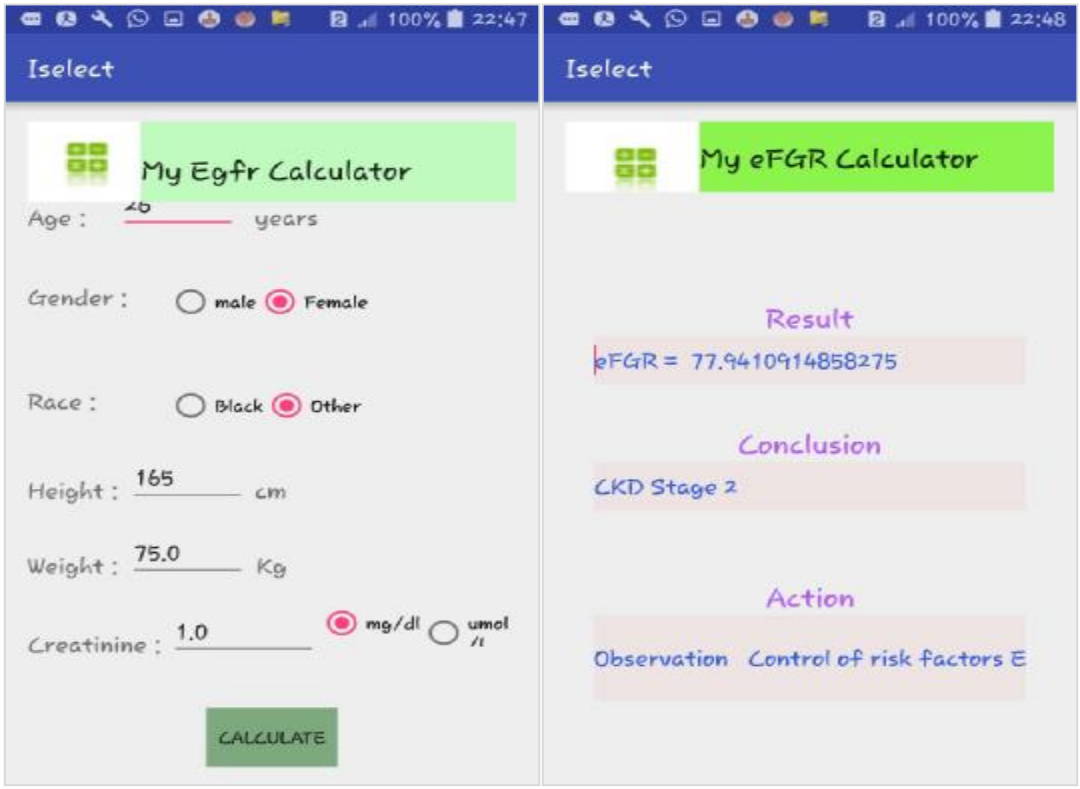
The development of the Arabic version of the application is still in progress.

2.1 Authentication pages:



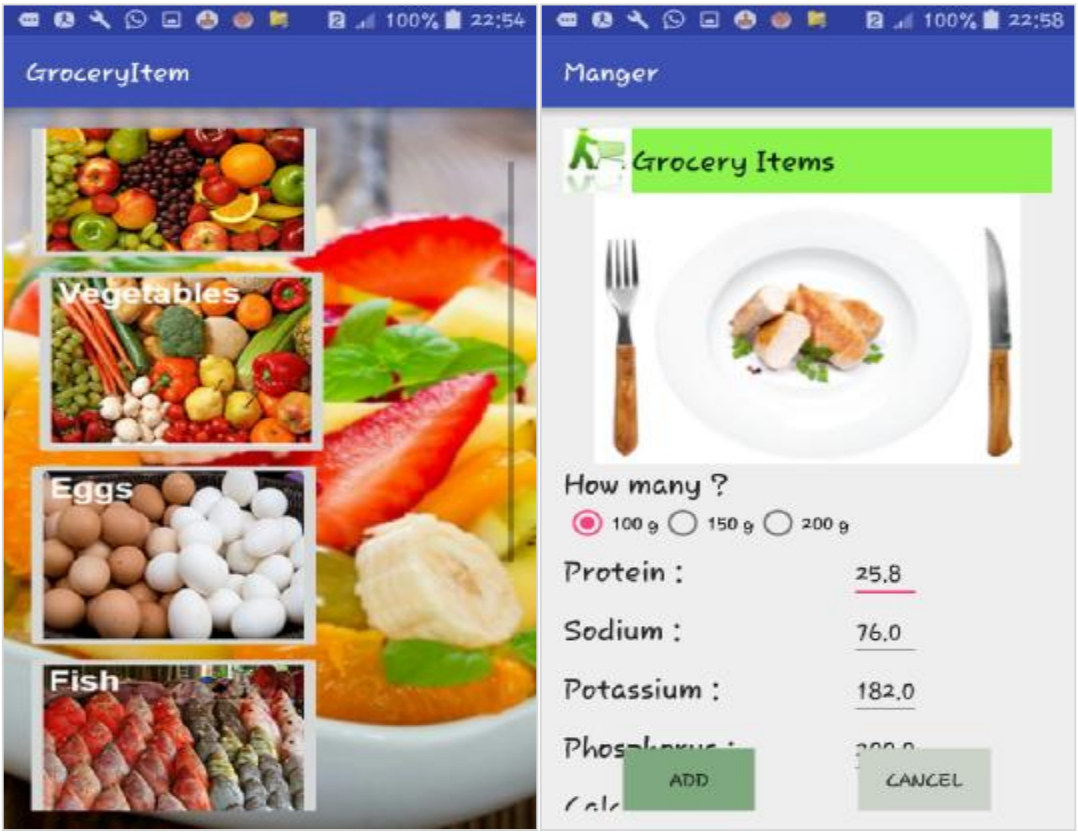
2.2 Personalize my profile:

2.3 My GFR calculator:

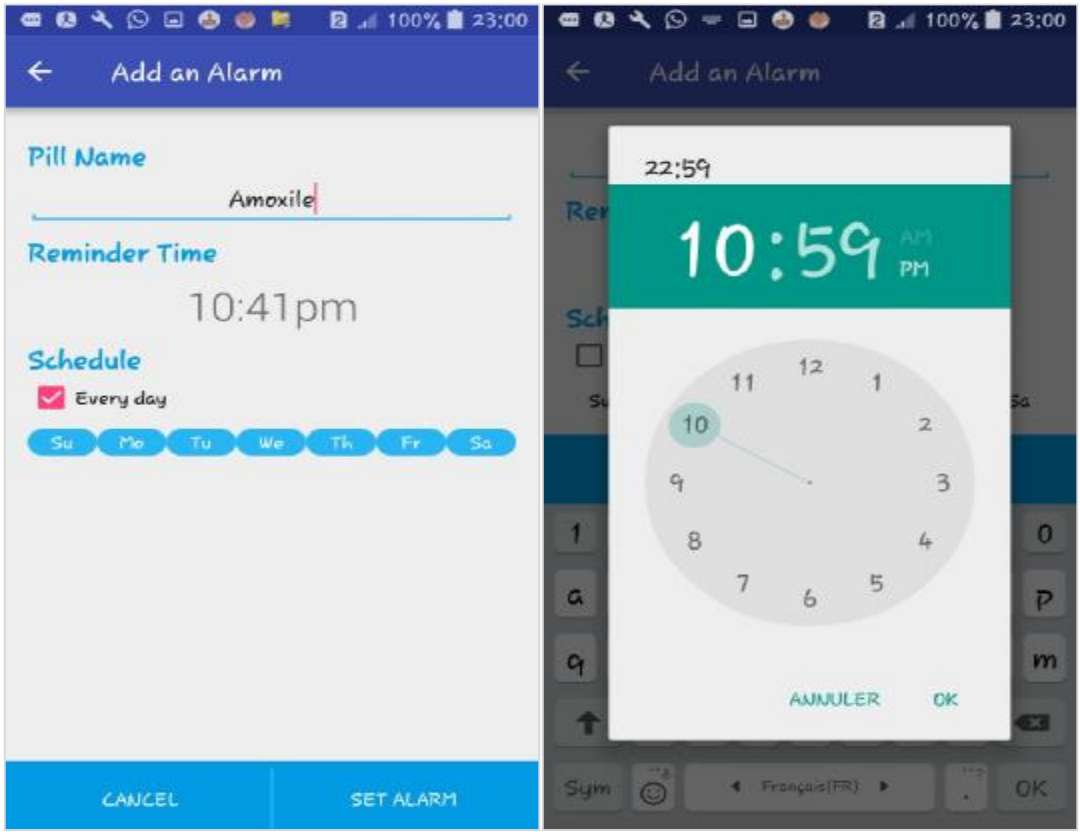


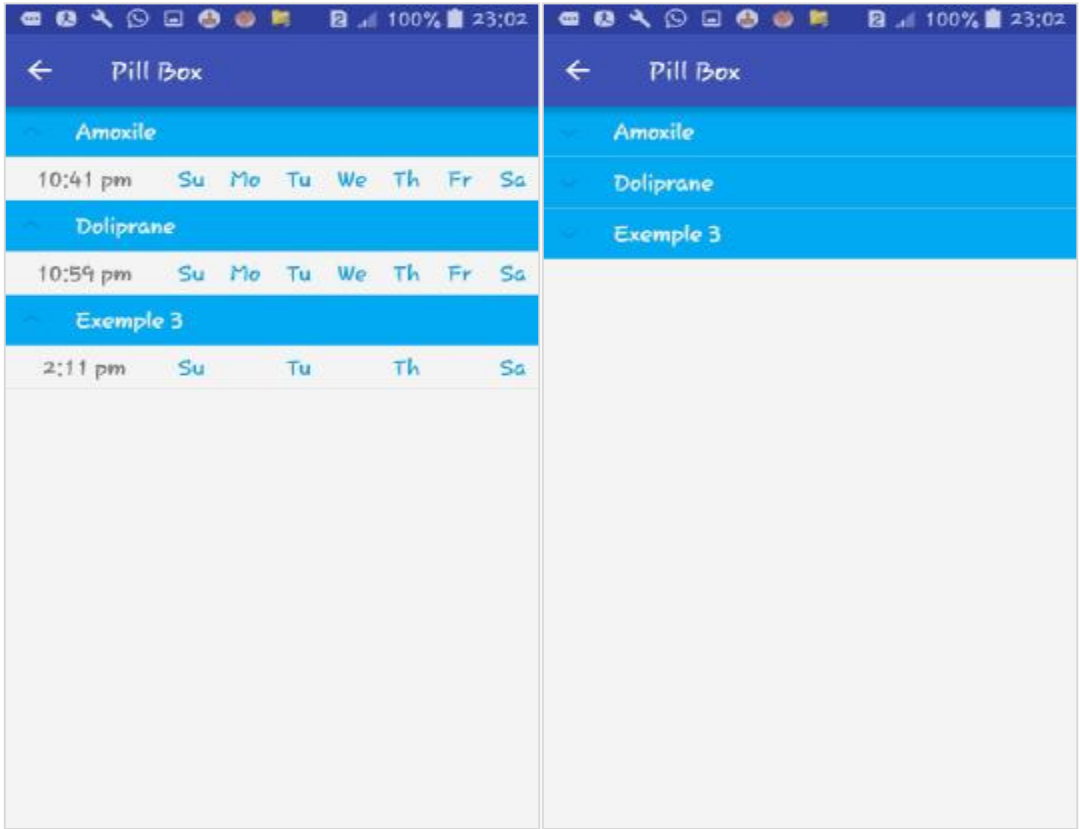
2.4 My dietary guidelines:

2.5 Grocery items:



2.6 MCIS:


Add an Alarm
Pill Name
Amoxile
Reminder Time
10:41pm
Schedule
Every day
Su Mo Tu We Th Fr Sa
CANCEL
SET ALARM
22:59
10:59 AM PM
ANNULER
OK

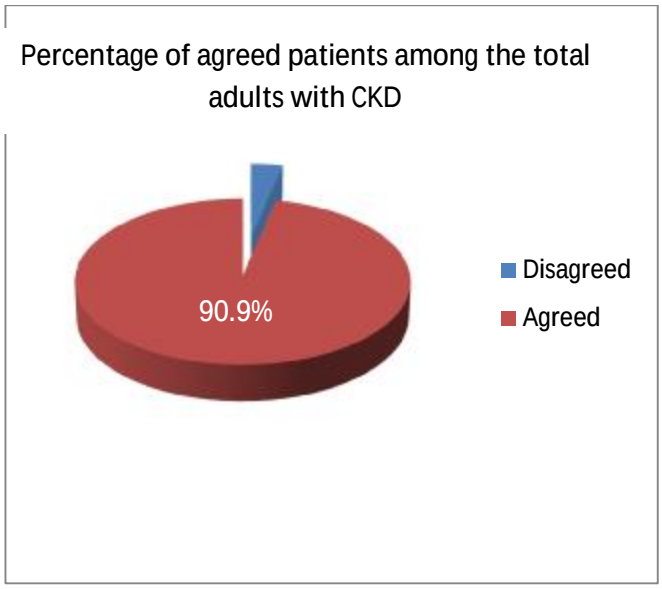

Pill Box
Amoxile
10:41 pm Su Mo Tu We Th Fr Sa
Doliprane
10:59 pm Su Mo Tu We Th Fr Sa
Exemple 3
2:11 pm Su Tu Th Sa
Amoxile
Doliprane
Exemple 3

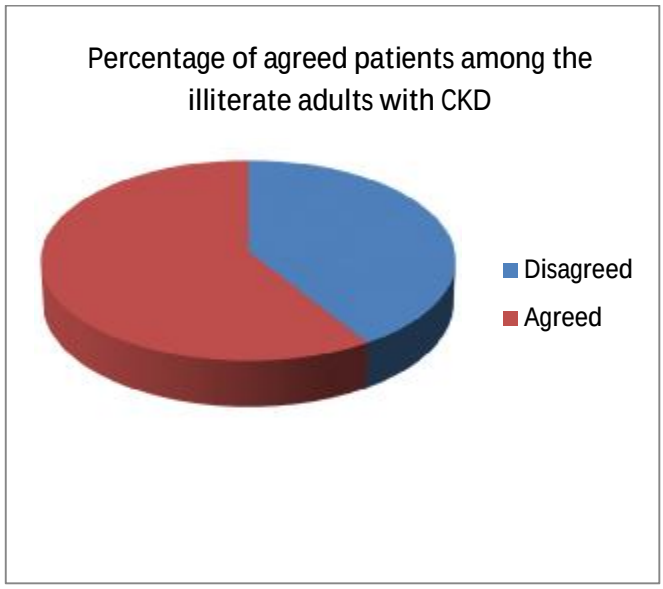
3.Survey's results:

- ü According to the questionnaire results, 90.9% of the interrogated adult patients with CKD, strongly agreed to use the application, either the Arabic or the English version.
- ü The main influencing factor among adult CKD patients, was the education level; in fact 41.1% of the illiterate adult patients refused the English version of the application, arguing that it will require more incorporation of the family members, which is in conflict with the main objective of the application (self-management).

Table 30: Distribution of adult patients with CKD according their education level

Adult CKD patients			
Educated		illiterate	
60		17	
Agreed	Disagreed	Agreed	Disagreed
60	0	10	7





- As regarding the questioned CKD children; 100% of them were eager to have the app on their Smartphones, claiming that they're ready to try any tool that could improve their life quality. The children parents were the key inciting factor.
- 7.21% of the total participants can't use a Smartphone (either they ignore how to use it or they can't afford it).
- 20.26% of participants had already experience with other self-management applications for CKD, but only 25% of them were satisfied.

DISCUSSION

Currently, the use of cutting edge technologies and particularly Smart home systems, has widely spread and became known to all of the world. In our country, likewise, the penetration rate of Smartphones' use is getting higher and higher yearly. In fact, the national agency of regulation of telecommunications (ANRT) has conducted at the beginning of 2016, an investigation about information & communication technologies which underscored that 94.4% of Moroccan people (12 to 65 years old) have a mobile phone, and that 54.7% of them are users of Smartphone. What is definitely encouraging is that 77.2% of those smartphone users, access into the internet in order to download applications.

Meanwhile, the faculty of medicine & pharmacy of Fez, follows the current national trends in matter of technologies. Indeed, in the last few years; lot of thesis projects of innovative technologies, have been realized. Notably; in neurology where many electronic systems have been created for syndromic diagnosis in neurology (2013), for the topographic diagnosis of peripheral neurogenic damage of the upper limb (2016), in addition to many interactive devices (CD ROM) for learning, self-education and assessment. Further to Serious Games' project which still in progress.

In parallel, the nephrology department brought forth; a social network for nephrology in Fez-Bouleman region (2011). Therapeutic education tools for patients in nephrology (2012). In addition to the E-Log book; a computer system for residents in nephrology (2014).

Likewise, our thesis comes to sit in the bottom of these evolutions. As a matter of fact, the major findings of this project and the interpretation of those findings are discussed in this chapter:

- Firstly the findings from the entire experience of designing iSelect application: Strong points of the app and its advantages of the platform in comparison with other nutrition applications for CKD, developed in other countries.

- Secondly, the different difficulties and challenges faced all along the development of the platform.
- thereafter, findings from the questionnaire, the several facts from verbal questions and answers will be discussed
- Then, how this application will impact both CKD patients and healthcare providers.
- Lastly this chapter will be concluded by discussing points to improve in the application, and the future work.

1. Strong points & advantages:

ü First Moroccan nutrition management application for patients with chronic kidney disease: This project is the first attempt to create a nutrition self-management mobile phone based application for Moroccan people with CKD. The menus' content, food items, recipes and all the database in the applications, were selected and brought from credible sources. As being the unique of its kind in morocco, iSelect will have a strong welcome from Moroccan CKD patients, especially that :

ü iSelect has special Moroccan particularities, since the nutritional content is adapted to Moroccan diet and eating habits, which are quite different from the other countries'. We can observe this through the varied meals integrated in the menu "recipes", but also through "Grocery items" menu, where all the usual Moroccan food items are present. This Moroccan touch makes useless, the nutrition applications for CKD available from other countries. Notably applications developed by National kidney Foundation (MY Food Coach App –

care after kidney transplant App – eGFR calculator App...), which are effective in some general options, but yet have specific food menu that could not be applied for all countries.(194)

- iSelect is “All in One”, it provides many options just on one plateform: A lot of systems have been developed in matter of CKD management, but each system was specifically designed to assure one or two particular functions. In other words- and as it is the case for NKF applications- calculating the eGFR requires downloading an application, nutrition self-management necessitates having another application, likewise, enhancing medical observance calls for a third application.(194) This fact of needing multitude of applications to ensure CKD nutrition management, make it non practical for patients, and is the reason why they break away from any related medical application.
- Language is the key to manipulate any system. In that regard, iSelect will be available in two versions. We opted for English since it is an international language and the most usable worldwide. iSelect could therefore have users from different regions in the world. However, an Arabic version is inevitably required, so as to make it accessible for all Moroccan patients despite of their educational level.

  This is an other strong point that privileges our application, compared with the other applications limited to French or English languages.
- Usability is what characterizes, iSelect, the most. As the design shows; There’s no place for complexities. Users could easily navigate on the app from a menu to an other by simple few clicks. Interfaces are clear enough and make patients directly in the objective.

2. Difficulties & challenges:

Even though, the overall design of iSelect application is positive, some difficulties were found as well. It can be summarized like below:

- The major challenge was to collect the database necessary for the applications. As Moroccan diet is so rich and diversified, it was difficult to collect all the large varieties of food items. This formed also an obstacle for iSelect encoding, since the more database is big, the more it requires free space on the app.
- The other difficulty found, is minimizing the information that could be displayed on each single interface. Some submenus on the application contain numerous informations and require then creation of more interfaces. In particular “Grocery items” menu.
- The application affords the micronutrients amounts contained in each 100mg of food product, which force users to weight food items all the time. This challenge seemed to be inevitable even with the auto recognition of photos, since in our Moroccan context; large ranges of size and weight does exist in food items notably in fruits and vegetables.
- The language that we should’ve build the application with, was an other challenge of the design process. As being the first foreign language in morocco; french was by far the suitable language for the application. However, with the intention of following the current Moroccan trends regarding language, and giving our application an international feature, we lastly opted for English as the official language of the App. Meanwhile, an Arabic version will be developed in order to make it more accessible for patients.

3. Finding from the questionnaire:

- ü All the participants answered that they want to have 'iselect' application, and that they were looking for such a programm. Except illiterate people who can't use Smartphone and could not have help from their relatives in this matter (7 patients), in fact they have suggested to make iSelect app usable on a normal phone.
- ü participants expressed that they want to try 'iSelect' for a longer period of time to observe how their body react on the food items they consume. Arguing that it would be very helpful for them to decide by themselves what they're about to eat.
- ü All the participants had no problem to weight food items. Their main concern were the accessibility (free application), and usability (ease to use).

4. iSelect application impact:

the use of iSelect application will be of great help not only for patients with chronic kidney failure, but also for nephrologists, residents and dietitians, seeing that it provides the possibility to control , monitor and assess the nutritional management of CKD on a hand, and to educate patients on the other hand.

As concerning healthcare providers; this application would be a pertinent tool to enhance the daily problems faced in the course of chronic kidney disease, particularly those related to nutritional management. Indeed, during medical consultations, doctors are supposed to educate CKD patients, convince them to change their eating habits, and teach them how to control their daily dietary intakes. Doctors have also the duty to inform patients about their CKD stage, their nutritional guidelines and make them aware about the potential complications they may have

later. And on top of all this, the doctors find it inescapable to propose a menu of some recipes and meals, for their patients. Unfortunately all these constraints require big efforts and take time, whence efficacy and outcomes are usually compromised. While within iSelect application, it will be easier to deal with nutrition management in CKD patients. Less effort and less time will be needed and better outcomes will be obtained, since medical consultations will focus on one single objective, is that of forming patients how to use the application, thereafter the application will take over the rest.

As chronic kidney disease is a chronic condition that requires chronic nutrition management and chronic follow-up, patients find it difficult to limit their food choices every day in order to reduce the nutrients intakes. Moreover, Moroccan diet make it even more hard for them to control protein and minerals inputs. iSelect application could be then considered as a virtual electronic doctor or dietitian who guides them all along the day in their diet. They will be able to make better food choices while keeping their intakes in the recommended interval. In addition, iSelect will be not only a self-management tool, but also a self-education mean, since it gives access to a simplified support where patients could learn more about their disease. Therefore, this application could significantly enhance outcomes in CKD management, especially that it helps further patients to ameliorate medical observation. And it is inescapable to note that the services offered by the platform will evidently impact positively, patients' quality of life, general health behavior and patient's beliefs about their illness.

iSelect does not uniquely impact doctors and patients, but either patient's family and relatives, who won't be forced anymore to incorporate in each step of

CKD management, since patients will self-manage their nutritional status by themselves.

5. Points to improve & future work:

More food items

More food items are encouraged to be included. This thesis project focused on the educational purpose of helping people with CKD to check by themselves and encourage them to change their food habits, besides the database is significantly huge. Therefore limited food items were included. However for the practical use, it would be much better if more items can be included.

History function

This system can save up unlimited items but does not provide a history function yet. The menu “My reports” that provide a history function to compare a person’s diet on 1 week or more is going to be included in ‘iSelect’ application.

Include activity measure function

There are other factors which can influence CKD. Physical activity is one of them. The function of tracing activities is also encouraged to incorporate.

Better way to measure food weight

Due to many constraints, the user of this system should weigh food items. If there is a way to estimate the eight of the food item without scale, notably auto photo recognition, it is recommended to be integrated into the application.

CONCLUSION

One of the most redoubtable medical issues, that have been widely spread and became known to all of the world, is that of Chronic kidney disease (CKD); this long-term condition where kidneys do not work normally any more, effects a large number of people whose diet should be adapted to their renal function, in order to prevent the renal disease worsening, on a hand, and avoid undernourishment on the other hand.

Throw *"iSelect"* application, we came to offer several services and options, to allow this category of patients, a free food items selection, on their own. While being reassured that it does correspond to their nutritional needs.

Using iSelect application in nephrology and particularly in nutritional management of chronic kidney disease, means that we opt for an important and practical system of monitoring, evaluation and education, but specially a system of self-management in one of the greatest challenges in CKD treatment that is control of nutrition status.

This platform which could be used in short or long term, privileges progress rather than the final result. It allows self-disease control and authentic assessment., improves patients beliefs and health behavior, and evidently contributes in enhancing patients' education while living with CKD.

ABSTRACT

Abstract

Background

Chronic kidney disease (CKD) is a worldwide health problem, affecting millions of people. It is defined as the presence of kidney damage with or without reduced kidney function.

The pathophysiology of chronic renal failure has helped to highlight the importance of dietary management of this disease, the fundamental role of monitoring by a dietician has now been clearly established. Efforts have been made to fully incorporate dietary intervention in the care of patients. It is no more about protein restriction only, but a full nutritional support must be offered. If it is not the case, patients with renal failure will be exposed to different nutritional risks. In fact, the protein and energy malnutrition is common in patients with CKD, and it increases with the function decline kidney. From where, development of new helpful tools is formally recommended.

Material and methods:

After several concertation meetings of the work team, have been organized at the nephrology department of Hassan 2 hospital, in order to set up a demarche for the realization of the project. In fact, it was decided that the Smartphone application will be developed according to the engineering approach described by Denning, which follows the processes: Design process, development process, implementation process and validation process.

In order to adapt more, our application to the users' requirements, we needed to study the target population, to assess their needs regarding the application, to figure out the challenges and constraints faced by CKD patients, and to bring out their own suggestions in matter of nutritional self-management in CKD. To achieve

those objectives, we opted for a survey methodology, to conduct among CKD patients.

ResuIts:

The design of the platform iSelect, has led to 3 main menus. The first menu contains 6 other submenus that allow to personalize the patient's profile, to estimate his/her GFR ,to afford daily dietary guidelines within nutrition reports, in addition to a further option of improving medical compliance. The second main menu gives access into inbox, share option, and education tips submenu where patients could learn about CKD.

As for the third menu, it allows to track nutrient intakes all along the day, but also to record any favorite recipe or meal that have been composed before.

Discussion:

iSelect application is the first nutrition self-management application for CKD patients that is made in Morocco. It has a lot of advantages compared to other similar platforms. In fact, it has many Moroccan particularities, it integrates several options and services on one single platform and it will be available in to languages.

The main challenges faced during the realization of the project, were the huge database that required more free interfaces and free space on the app, in addition to minimizing the information displayed and the way to measure food weight.

However, iSelect app still a beneficial tool that impact positively, not only patients and healthcare providers, but also patient's family and relatives. It allows enhancing life quality of patients and CKD management outcomes, with less efforts and less time.

RESUME

Introduction:

L'insuffisance rénale chronique (IRC) est un problème de santé publique, affectant des millions de personnes dans le monde entier. Il se définit comme étant la dégradation progressive et irréversible de la fonction rénale. La physiopathologie de l'insuffisance rénale chronique a permis de mettre en évidence l'importance de la prise en charge diététique dans cette maladie, le rôle fondamental de la surveillance par un diététicien a été clairement établi. Des efforts ont été faits pour intégrer pleinement l'intervention diététique dans la prise en charge des patients, en effet il ne s'agit plus d'une simple restriction protéique seulement, mais tout un soutien nutritionnel complet doit être offert. Dans le cas contraire, les patients souffrant d'insuffisance rénale chronique seront exposés à différents risques nutritionnels dont la malnutrition proteino-énergetique qui est assez fréquente chez les patients atteints d'IRC, et dont le risque augmente avec la dégradation de la fonction rénale. Le développement de nouveaux outils et systèmes pour améliorer la PEC nutritionnelles est donc fortement recommandé.

Matériels et méthodes :

Après plusieurs réunions de concertation de l'équipe de travail, qui ont été organisé au service de néphrologie de l'hôpital Hassan 2, afin de mettre en place une démarche pour la réalisation du projet, il a été décidé que l'application Smartphone sera développée selon l'approche d'ingénierie décrite par Denning, qui suit 4 processus: processus de conception, processus de développement, le processus d'implantation et le processus de validation.

Afin d'adapter encore plus, notre application aux besoins des utilisateurs, nous avions besoin d'étudier la population cible, afin d'évaluer leurs besoins en ce

qui concerne l'application, de comprendre les défis et les contraintes rencontrées par cette catégorie de patients, et de faire ressortir leurs propres suggestions en ce qui concerne l'autogestion du statut nutritionnel au cours de l'IRC. Afin d'atteindre ces objectifs, nous avons opté pour la réalisation d'un sondage chez les patients atteints d'IRC.

Résultats :

La conception de la plate-forme iSelect, à mener à la création de 3 menus principaux. Le premier menu contient 6 autres sous-menus qui permettent, de personnaliser le profil du patient, d'estimer son débit de filtration glomérulaire, d'informer le patient sur ses besoins nutritionnels quotidiens, de fournir des rapports sur les repas choisis dans les jours précédents, mais également d'améliorer l'observance médicale. Le second menu principal donne accès à la boîte de réception, à une option de partage des informations personnelles, et aussi à un support éducatif où les patients pourraient apprendre davantage sur leur maladie.

En ce qui concerne le troisième menu, il permet de surveiller les apports nutritionnels tout au long de la journée, mais aussi d'enregistrer toute recette ou repas préféré qui ont déjà été composées et validés avant.

Discussion :

l'applicationiSelect est la première application d'autogestion de la nutrition pour les patients atteints d'IRC, développée au Maroc. Elle présente beaucoup d'avantages par rapport à d'autres plates-formes similaires. En fait, elle offres plusieurs particularités marocaines, il intègre plusieurs options et services sur une seule plate-forme et il sera disponible en 2 versions, anglaise et arabe.

Le principal défi rencontré au cours de la réalisation du projet, était l'énorme base de données qui exigeait plus d'espace libre et la création de plus d'interfaces sur l'application. Ajoutant à ceci le challenge de minimiser les informations qui

peuvent être affichées sur un seul interface, et la difficulté de faire un choix d'alimentes , qui n'est pas basé sur la mesure de leur poids.

Mais tout de même, iSelect reste un outil fiable qui a un impact positif, non seulement sur les patients et les médecins, mais aussi sur la famille et les proches du patient. Il permet d'améliorer le pronostic et la qualité de vie des patients avec IRC, avec un minimum d'effort et un gain de temps.

ملخص

المقدمة

يعد القصور الكلوي المزمن, أو ما يعرف كذلك بالفشل الكلوي المزمن, من أكبر مشاكل الصحة العامة في العالم, ويعرف هذا القصور كحالة مرضية تتصف بعجز الكلى عن أداء وظائفها بشكل طبيعي. وقد أثبتت الدراسات أن للتغذية المتوازنة دور فعال و أساسي في إطار علاج هذا المرض. إذ أصبحت المتابعة مع اختصاصي التغذية أمر مفروغ من أهميته. ولقد تم القيام بمحاولات جد كبيرة من أجل إدماج التغذية كمحور أساسي من محاور علاج المرض, فلم تعد المتابعة بالتوجيهات الغذائية تقتصر على النقص من كمية البروتينات فقط, بل هناك دعم كامل وتوصيات غذائية شاملة تتم مراقبتها. وعلى العكس من ذلك فإن التغذية غير المتوازنة تعرض أصحابها لمجموعة من الآفات كسوء التغذية التي تعتبر حالة صحية متفشية بين المرضى المصابين بالفشل الكلوي المزمن. ومنه, فإن ابتكار وخلق وسائل و أنظمة جديدة لتسهيل التحكم في النظام الغذائي لهذه الفئة من المرضى, أصبح أمرا ضروريا.

الأساليب

بعد العديد من الاجتماعات مع فريق العمل بمصلحة أمراض الكلى بالمركز الاستشفائي الحسن الثاني, تم اتخاذ قرار تحقيق هذا المشروع و تطوير التطبيق Select وفقا لمقاربة هندسية التي وصفها دينين والتي تستوجب 4 عمليات: عملية التصميم, عملية البرمجة, عملية الدمج, ثم عملية التقييم.

ومن أجل استيعاب احتياجات مستعملي هذا التطبيق بشكل أوضح, ارتأينا الى القيام باستطلاع الرأي موجه لمرضى القصور الكلوي, من أجل دراسة هذه الفئة المستهدفة, وتقييم احتياجاتها من التطبيق و كذلك من أجل معرفة مختلف التحديات والصعوبات التي تواجه هذا النوع من المرضى في محاولتهم للتحكم في نظامهم الغذائي.

النتائج

قمنا بتصميم التطبيق, ثم الحصول على ثلاث واجهات رئيسية, حيث تضم الواجهة الرئيسية الاولى 6 واجهات أخرى, تمكن من تعديل الملف الشخصي لكل مستعمل, ثم حساب مؤشر الكتلة الجسمية, منح المريض نصائح حول التوجيهات الغذائية, تخطيط الوجبات اليومية, بالاضافة الى وصفات صحية وتقارير عن النظام الغذائي الذي تم اتباعه في الايام الفارطة. بينما تمكن الواجهة الرئيسية الثانية من الولوج الى علبة الرسائل, من نشر ومشاركة المعلومات الخاصة بالمريض مع باقي المستخدمين و كذلك من الولوج الى حيز خاص من التطبيق حيث يمكن للمريض أن يقرأ و يتعرف أكثر على مرضه. أما بالنسبة للواجهة الرئيسية الثالثة فهي تخول تتبع كميات العناصر المعدنية والبروتينات المستهلكة خلال اليوم و كذلك القيام بحفظ أي وصفة تم اختيارها من قبل.

التحاليل

يعتبر iSelect أول تطبيق هاتفي يتم ابتكاره في المغرب, في اطار ادارة النظام الغذائي لدى مرضى القصور الكلوي. يمتلك هذا التطبيق العديد من الفوائد التي تميزه عن غيره من التطبيقات, اذ أنه يوفر مجموعة من الخدمات الخاصة ذات طابع مغربي, كما أنه يقدم مجموعة من الوظائف و الخيارات على نفس التطبيق, و ذلك بلغتين مختلفتين: العربية والانجليزية. ومن أهم التحديات التي واجهتنا أثناء تحقيق هذا المشروع, الا وهو حجم

المعطيات التي يجب إدخالها في التطبيق، بالإضافة إلى تحديد كمية المعلومات التي يمكن أن تظهر على كل واجهة، وكذلك اختيارات المستخدم للمواد الغذائية التي تستوجب القيام بوزن هذه الأغذية. ومع هذا كله، يظل هذا التطبيق مهما ليس فقط بالنسبة للمرضى القصور والأطباء بل وكذلك بالنسبة للأسرة وأقارب المريض، إذ أنه يطور وجود حياة المرضى، يحسن من التزامهم الغذائي ويحسن من نتائج العلاج.

REFERENCES

(1) World Kidney Day: Chronic Kidney Disease. 2015; http://www.worldkidneyday.org/faqs/chronic-kidney-disease

(2) ANZDATA registry report 2005, in Australia and new Zealand dialysis and transplant registry. 2006: Adelaide, south Australia.

(3) Saydah S, Eberhardt M, Rios-burrows N, Williams D, Geiss L, and Dorsey R: prevalence of chronic kidney disease and Associated risk factors-united states, 1999-2004. MMWR Morb Mortal Wkly Rep 56: 161-165, 2007

(4) Centers for Disease Control and Prevention. Chronic Kidney Disease Surveillance System. Atlanta. Atlanta, GA: Centers for Disease Control and Prevention, US Dept of Health and Human Services; 2011. http://www.cdc.gov/ckd. Accessed August 5, 2013.

(5) National Kidney Foundation Practice Guidelines for Chronic Kidney Disease: Evaluation, Classification, and Stratification. From Tufts-New England Medical Center and Tufts University School of Medicine, avaibale on the following likn: http://annals.org/article.aspx?articleid=716575

(6) Levey AS, Coresh J, Balk E, Kausz AT, Levin A, Steffes MW, et al. National Kidney Foundation practice guidelines for chronic kidney disease: evaluation, classification, and stratification. Ann Intern Med. 2003;139(2):137-47

(7) Heimburger O, Qureshi AR, Blaner WS, Berglund L, Stenvinkel P. Hand-grip muscle strength, lean body mass, and plasma proteins as markers of nutritional status in patients with chronic renal failure close to start of dialysis therapy.Am J Kidney Dis. 2000;36(6):1213-25.

(8) Qureshi AR, Alvestrand A, Danielsson A, Divino-Filho JC, Gutierrez A, Lindholm B, et al. Factors predicting malnutrition in hemodialysis patients: a crosssectional study. Kidney Int. 1998;53(3):773-82.

(9) Young GA, Kopple JD, Lindholm B, Vonesh EF, De Vecchi A, Scalamogna A, et al. Nutritional assessment of continuous ambulatory peritoneal dialysis patients: an international study. Am J Kidney Dis. 1991;17(4):462-71.

(10) Carrero JJ, Stenvinkel P, Cuppari L, Ikizler TA, Kalantar-Zadeh K, Kaysen G, et al. Etiology of the protein-energy wasting syndrome in chronic kidney disease: a consensus statement from the International Society of Renal Nutrition and Metabolism (ISRNM). J Ren Nutr. 2013;23(2):77-90

(11) Kovesdy CP. Traditional and novel dietary interventions for preventing progression of chronic kidney disease. J Ren Nutr. 2013;23(3):241-5

(12) de Brito-Ashurst I, Varagunam M, Raftery MJ, Yaqoob MM. Bicarbonate supplementation slows progression of CKD and improves nutritional status. J Am SocNephrol. 2009;20(9):2075-84

(13) Hagger MS, Orbell S. A meta-analytic review of the common-sense model of illness representations. Psychology & Health. 2003 Apr;18(2):141-84.

(14) Jansen DL, Grootendorst DC, Rijken M, Heijmans M, Kaptein AA, Boeschoten EW, et al. Pre-dialysis patients' perceived autonomy, self-esteem and labor participation

(15) Gartner Identifies the Top 10 Consumer Mobile Applications for 2012. Available at: http://www.gartner.com/newsroom/id/1230413, accessed August 2013

(16) The Kidneys and How They Work. *National Kidney and Urologic Diseases Information Clearinghouse (NKUDIC)*

(17) A Brief Atlas of the Human Body, http://www.phschool.com/atschool/florida/pdfbooks/sci_Marieb/pdf/Marieb_ch25.pdf

(18) http://www.innerbody.com/image_urinov/dige05-new.html#full-description

(19) Wein AJ, Kavoussi LR, Novick AC, Partin AW, Peters CA. *Campbell-Walsh Urology*. 9th Ed. Saunders; 2007. Vol I: p 24.

(20) http://www.aboutcancer.com/kidney_anatomy.htm

(21) Robbins and Cotran Pathologic Basis of Disease 7E

(22) K/DOQI clinical practice guidelines for chronic kidney disease : Evaluation, classification and stratification. Kidney disease outcome quality initiative (NKF KDOQI)tm , the national kidney foundation avaibale from : http://www.kidney.org/professionals/KDOQI/guidelines ckd/toc.htm

(23) National Kidney Foundation. K/DOQI clinical practice guide- lines for chronic kidney disease: evaluation, classification, and stratification. Am J Kidney Dis 39(2 suppl 1) : S1-266,2002

(24) Caillard B, Durand J, et Verret J. Antibiotherapy and renalinsuffisancy. Anesthésie, Analgésie, Réanimation. Masson Et Cie. ParisFrance 197

(25) ANAES. Diagnostic de l'insuffisance rénale chronique chez l'adulte; 2002

(26) Simon P. L'insuffisance rénale : prévention et traitements. Elsevier Masson S.A.S; 2007

(27) World Kidney Day: Chronic Kidney Disease. 2015; http://www.worldkidneyday.org/faqs/chronic-kidney-disease/ .

(28) Jha V, Garcia-Garcia G, Iseki K, et al. Chronic kidney disease: global dimension and perspectives.*Lancet.* Jul 20 2013;382(9888):260-272

(29) Levey AS, Atkins R, Coresh J, et al. Chronic kidney disease as a global public health problem: approaches and initiatives - a position statement from Kidney Disease Improving Global Outcomes.*Kidney Int.* Aug 2007;72(3):247-259.

(30) Couser WG, Remuzzi G, Mendis S, Tonelli M. The contribution of chronic kidney disease to the global burden of major noncommunicable diseases. *Kidney Int.* Dec 2011;80(12):1258-1270.

(31) Kidney Int. 2016 Jun;89(6):1363-71. doi: 10.1016/j.kint.2016.02.019. Epub 2016 Apr 13. Chronic kidney disease, hypertension, diabetes, and obesity in the adult population of Morocco: how to avoid "over"- and "under"-diagnosis of CKD.

(32) BenghanemGharbi M. Elseviers M. Zamd M. BelghitiAlaoui A. Benhadi N. Trabelssi El. Bayahia R. Ramdani B. E. de Broe M. Chronic kidney disease, hypertension, diabetes and obesity in the adult population of Morocco: How to ovoid over and under diagnosis of CKD. Kidney international. 2016; 9:5-7

(33) Therapeutic education of adults with advanced CKD .thesis made and presented ON 29/06/2012 BY Mr. YunusAnsisse in the faculty of medicine and pharmacy of Fez.

(34) Eckardt KU, Coresh J, Devuyst O, Johnson RJ, Kottgen A, Levey AS, Levin A: Evolving importance of kidney disease: from subspecialty to global health burden. Lancet 2013, 382(9887):158-169

(35) Coresh J, Selvin E, Stevens LA, Manzi J, Kusek JW, Eggers P, Van Lente F, Levey AS: Prevalence of chronic kidney disease in the United States. JAMA : the journal of the American Medical Association 2007, 298(17):2038-2047

(36) Levey AS, Coresh J, Balk E, Kausz AT, Levin A, Steffes MW, Hogg RJ, Perrone RD, Lau J, Eknoyan G et al: National Kidney Foundation practice guidelines for chronic kidney disease: evaluation, classification, and stratification

(37) KDOQI clinical Practice Guidelines for CKD: Evaluation, classification and stratification. Kidney disease outcome quality initiative (NKF- KDOQI)™ , the nationa kidney foundation. Avaibale from : www.kidney.org/professionals/kdoqi/guidelines_ckd/toc.htm

(38) Uhlig K, Levy AS, Sarnak MJ. Traditional cardiac risk factors in individuals with CKD. SEmin Dial 16(2): 118-127, 2003.

(39) Sarnak MJ, levy AS. Cardiovascular disease and chronic kidney disease: a new paradigm. Am J kidney Dis 35(4 suppl):S117-131, 2000

(40) Measurement of Kidney Function Lesley A. Stevens, MD, FRCPC*, Andrew S. Levey, MD Division of Nephrology, Tufts University School of Medicine, Tufts–New England Medical Center, 750 Washington Street, Box 391, Boston, MA 02111, USA.

(41) N Am J Med Sci. 2010 Apr; 2(4): 170–173. Markers of renal function tests ShivarajGowda, Prakash B. Desai, Shruthi S. Kulkarni, Vinayak V. Hull, Avinash A. K. Math, andSonal N. Vernekar

(42) Yuegang Z, Chengjun W, et al. Simultaneous Determination of Creatinine and Uric Acid in Human Urine by High Performance Liquid Chromatography. Anal Sci. 2008;24:1589–1592.[PubMed]

(43) Corbett JV. 7th Ed. 2008. Laboratory tests and diagnostic procedures with nursing diagnoses; pp. 90–107.

(44) Miller W, Myers G, Ashwood E, et al. Creatinine measurement: state of the art in accuracy and interlaboratory harmonization. Arch Pathol Lab Med. 2005;129(3):297–304. [PubMed]

(45) Banfi G, Del F. Serum creatinine values in elite athletes competing in 8 different sports: comparison with sedentary people. Clin Chem. 2006;52:330–331. [PubMed]

(46) Branten AJ, Vervoort G, Wetzels JF. Serum creatinine is a poor marker of GFR in nephrotic syndrome. Nephrol Dial Transplant. 2005;20:707–711. [PubMed]

(47) Edmund L, David J. Kidney function tests. In: Carl AB, Edward R, David E, editors. Tietz Textbook of clinical chemistry and molecular diagnostics. 4th ed. New Delhi: Elsevier Inc; 2006. pp. 797–808

(48) Mitchell HR and Kline W. Core curriculum in nephrology, Renal Function Testing. Am J Kidney Dis. 2006;47:174–183. [PubMed]

(49) Pagana , Kathleen D. Mosby's Manual of Diagnostic and Laboratory Tests. St. Louis Mosby, Inc.1998 and Rebecca J.F Gale Encyclopedia of Medicine. 2002

(50) Dharnidharka VR, Kwon C, Stevens G. Serum cystatin C is superior to serum creatinine as a marker of kidney function: a meta-analysis. Am J KidneyDis. 2002;40:221–226.[PubMed]

(51) Cholongitas E, Shusang V, Marelli L, et al. Review article: renal function assessment in cirrhosis - difficulties and alternative measurements. Aliment Pharmacol Ther.2007;26(7):969–978. [PubMed]

(52) Gerbes AL, Gulberg V, Bilzer M, et al. Evaluation of serum cystatin C concentration as a marker of renal function in patients with cirrhosis of the liver. Gut. 2002;50:106–110.[PMC free article] [PubMed]

(53) Laura P, Stefano T, Daniela L, et al. Cystatin C and Estimates of Renal Function: Searching for a Better Measure of Kidney Function in Diabetic Patients. Clin Chem.2007;53:480–488. [PubMed]

(54) Shlipak MG, Katz R, Fried LF, et al. Cystatin-C and mortality in elderly persons with heart failure. J Am CollCardiol. 2005;45:268–271. [PubMed]

(55) riem F, Althaus H, Birnbaum M, et al. ß-trace protein in serum: a new marker of glomerular filtration rate in the creatinine-blind range. Clin Chem. 1999;45:567–568. [PubMed]

(56) Woitas RP, Stoffel-Wagner B, Poege U, et al. Low-molecular weight proteins as markers for glomerular filtration rate. Clin Chem. 2001;47:2179–2180. [PubMed]

(57) Hoffmann A, Nimtz M, Conradt H. Molecular characterization of ß-trace protein in human serum and urine: a potential diagnostic marker for renal diseases. Glycobiology. 1997;7:499–506. [PubMed]

(58) Priem F, Althaus H, Jung K, Sinha P. ß-Trace protein is not better than cystatin C as an indicator of reduced glomerular filtration rate. Clin Chem. 2001;47:2181. [PubMed]

(59) Jacobsen LA. Method for calculation of renal clearance based upon a single plasma sample. Clin Physiol. 1983;3:297–305. [PubMed]

(60) Physiology: 7/7ch04/7ch04p11 - Essentials of Human Physiology - "Glomerular Filtration Rate"

(61) Cockcroft DW, Gault MH (1976). "Prediction of creatinine clearance from serum creatinine". *Nephron*. 16 (1): 31–41.doi:10.1159/000180580. PMID 1244564.

(62) *Gault MH, Longerich LL, Harnett JD, Wesolowski C (1992). "Predicting glomerular function from adjusted serum creatinine". Nephron. 62 (3): 249–56.*doi:10.1159/000187054. PMID 1436333.

(63) "GFR MDRD Calculator for Adults". *National Kidney Disease Education Program. United States:* National Institutes of Health.

(64) *Hougardy, JM; Delanaye, P; Le Moine, A; Nortier, J (2014). "Estimation of the glomerular filtration rate in 2014 by tests and equations: strengths and weaknesses.". Rev Med Brux. (in French). 35 (4): 250–7.* PMID 25675627.

(65) Schwartz GJ, Haycock GB, Edelmann CM, Spitzer A (August 1976). "A simple estimate of glomerular filtration rate in children derived from body length and plasma creatinine". *Pediatrics*. 58 (2): 259–63. PMID 951142.

(66) *Schwartz GJ, Feld LG, Langford DJ (June 1984). "A simple estimate of glomerular filtration rate in full-term infants during the first year of life". The Journal of Pediatrics. 104 (6): 849–54.* doi:10.1016/S0022-3476(84)80479-5. PMID 6726515.

(67) Brion LP, Fleischman AR, McCarton C, Schwartz GJ (October 1986). "A simple estimate of glomerular filtration rate in low birth weight infants during the first year of life: noninvasive assessment of body composition and growth".*The Journal of Pediatrics*. 109 (4): 698–707.

(68) Nationa kidney foundation , glomerular filtration rate, avaibale on https://www.kidney.org/atoz/content/gfr

(69) "United States Renal Data System (USRDS)".

(70) Rahman, Mahboob; Smith, Michael C. (September 1998). *"Chronic renal insufficiency: A diagnostic and therapeutic approach"*. Archives of Internal medicine. 158: 1743–52. *doi*:*10.1001/archinte.158.16.1743*. *PMID 9738603*.

(71) SeminNephrol. 2002 Jan;22(1):17-26.Pathophysiology of hypertension in renal failure.

(72) Annu Rev Pathol. 2011;6:395-423. doi: 10.1146/annurev.pathol.4.110807.092150.A glimpse of various pathogenetic mechanisms of diabetic nephropathy.

(73) SeminNephrol. 2007 Mar;27(2):130-43.AGE, RAGE, and ROS in diabetic nephropathy.

(74) SeminNephrol. 2007 May;27(3):321-37.The many effects of complement C3- and C5-binding proteins in renal injury.

(75) J Am SocNephrol. 2008 Apr;19(4):651-3. doi: 10.1681/ASN.2008020156. Epub 2008 Mar 5.Are podocytes passive or provocative in proteinuric glomerular pathology?

(76) J Am SocNephrol. 2003 Jul;14(7 Suppl 2):S186-91. Chronic progression of tubulointerstitial damage in proteinuric renal disease is mediated bycomplement activation: a therapeutic role for complement inhibitors?

(77) BMJ. 2002 Jul 13; 325(7355): 85–90.PMCID: PMC1123635Chronic renal disease

(78) OuattaraB, OuffoueKra, Hubert Y, Kadjo K, EzaniKodjo N : Particularités de l'insuffisance rénale chronique chez des patients adultes noirs hospitalisés dans le service de médecine interne du CHU de Treichville, nephro.2011.03.009

(79) EyoussfiS: Néphropathie diabétique lors de la première consultation. Thèse n° 029 /2011. Faculté de médecine et de pharmacie. Fès

(80) HannedouchT : Néphropathie vasculaires: généralitésetclassification.WWW. nephrohus.org

(81) AlainM : Athérosclérose et reins.2005.06.006.

(82) Conlon PJ, Little MA, Pieper K, Mark DB : Severity of renal vascular disease predicts mortality in patients undergoing coronary angiography. Kidney Int 2001;60:1490–7.

(83) BeaufilsM :Néphroangiosclérose. EMC-Néphrologie 2 (2005) 103–124

(84) Frimat L, Loos-Ayav C, Briançon S, Kessler M : Épidémiologie des maladies rénales chroniques. EMC-Néphrologie 2 (2005) 139–157.

(85) Sotirakapoilos N, Tsitsios T, Mstanbolidou C, SparaS :Mavromatidis anticipation ofend stage renal diseas in patients with autosomal dominant polycystic kidney diseas in successive generations. Renal failure 2001; 23:715-720

(86) Marie-Claire G, Laurence H, CorinneA: Syndrome d'Alport ou néphropathie héréditaire hématurique progressive avec surdité ; Néphrologie & Thérapeutique 3 (2007) 113–120

(87) Eddy AA Progression in chronic kidney disease. Adv Chronic Kidney Dis2005;12:353-365.

(88) Yu HT Progression of chronic renal failure. Arch Intern Med 2003;163:1417-1429.

(89) How does early chronic kidney disease progress? A Background Paper prepared for the UK Consensus Conference on Early Chronic Kidney Disease. © The Author [2007]. Published by Oxford University Press on behalf of ERA-EDTA

(90) Buckalew VM, Berg RL, Wang SR, et al Prevalence of hypertension in 1,795 subjects with chronic renal disease: the modification of diet in renal disease study baseline cohort. Modification of Diet in Renal Disease Study Group. Am J Kidney Dis1996;28:811-821.

(91) Ruiz-Ortega M, Esteban V, Ruperez M, et al Renal and vascular hypertension-induced inflammation: role of angiotensin II. Current Opinion Nephrol Hypert2006;15:159-166

(92) . Perico N, Codreanu I, Schieppati A, Remuzzi G Pathophysiology of disease progression in proteinuric nephropathies. Kidney Int 2005;67:S79-S82.

(93) Nankivell BJ, Boadle RA, Harris DC Iron accumulation in human chronic renal disease. Am J Kidney Dis 1992;20:580-584.

(94) The GISEN group. Randomised placebo-controlled trail of effect of ramipril on decline in glomerular filtration rate and risk of terminal renal failure in proteinuric, non-diabetic nephropathy. Lancet 1997;349:1857-1863.

(95) Author manuscript; available in PMC 2009 Jun 1. Published in final edited form as: Prim Care. 2008 Jun; 35(2): 329–vii.doi: 10.1016/j.pop.2008.01.008. Chronic kidney disease and its complications.

(96) Besarab A, Levin A. Defining a renal anemia management period. Am.J.Kidney Dis.2000;36:S13–S23. [PubMed]

(97) McClellan W, Aronoff SL, Bolton WK, et al. The prevalence of anemia in patients with chronic kidney disease. Curr.Med.Res.Opin. 2004;20:1501–1510. [PubMed]

(98) McClellan W, Aronoff SL, Bolton WK, et al. The prevalence of anemia in patients with chronic kidney disease. Curr.Med.Res.Opin. 2004;20:1501–1510.

(99) Ratcliffe PJ. Molecular biology of erythropoietin. Kidney Int. 1993;44:887–904.

(100) Clinical epidemiology of cardiovascular disease in chronic renal disease.*Foley RN, Parfrey PS, SarnakMJAm J Kidney Dis. 1998 Nov; 32(5 Suppl 3):S112-9.*

(101) Kasiske BL. Hyperlipidemia in patients with chronic renal disease. American Journal of Kidney Diseases. 1998;32:S142–S156

(102) The hyperlipidemia of the nephrotic syndrome. Relation to plasma albumin concentration, oncotic pressure, and viscosity.*Appel GB, Blum CB, Chien S, Kunis CL, Appel AS N Engl J Med. 1985 Jun 13; 312(24):1544-8.*

(103) Death during the first 90 days of dialysis: a case control study.*Khan IH, Catto GR, Edward N, MacLeod AM Am J Kidney Dis. 1995 Feb; 25(2):276-80.*

(104) Nat Rev Nephrol. 2011 Mar;7(3):128-30. doi: 10.1038/nrneph.2010.168. Epub 2010 Dec 21.Chronic kidney disease: blood-pressure targets in chronic kidney disease

(105) Nephrol Dial Transplant. 2002 May;17(5):723-31. Management of disturbances of calcium and phosphate metabolism in chronic renal insufficiency, with emphasis on the control of hyperphosphataemia.

(106) Lancet. 2012 Jan 14;379(9811):165-80. doi: 10.1016/S0140-6736(11)60178-5. Epub 2011 Aug 15. Chronickidneydisease

(107) Hand L. Antihypertensives May Delay Kidney Disease Progression. *MedscapeMedical News*. Dec 16 2013

(108) Hsu TW, Liu JS, Hung SC, et al. Renoprotective effect of renin-angiotensin-aldosterone system blockade in patients with predialysis advanced chronic kidney disease, hypertension, and anemia. *JAMA Intern Med*. 2013 Dec 16.

(109) Park M, Hsu CY. An ACE in the hole for patients with advanced chronic kidney disease?. *JAMA Intern Med*. 2013 Dec 16.

(110) Chronic Kidney Disease Treatment & Management, Pradeep Arora, MD Assistant Professor of Medicine, University of Buffalo State University of New York School of Medicine and Biomedical Sciences; Attending Nephrologist, Veterans Affairs Western New York Healthcare System

(111) Audrey Berthet. Nutrition et insufisancerenale chronique. Sciences pharmaceutiques. 2009.dumas-01165273>

(112) Carr S. Renal replacement therapy for diabetic nephropathy, in Johnson RJ and Feehally J (eds): Comprehensive Clinical Nephrology, Mosby, London, 2000, pp 36.1-36.10.

(113) http://www.slideshare.net/phicna2005/hemodialysis-unit

(114) National kidney foundation. About peritoneal dialysis, avaibale onhttp://www.nkfs.org/treatment/peritoneal-dialysis/about-peritoneal-dialysis/

(115) Registre national de l'insuffisance rénale chronique terminal Magredial Premier rapport annuel. Edition 2009. (Ministère de la santé, direction des Hôpitaux et des soins Ambulatoires –Société Marocaine de NéphrologieAssociation des Néphrologues au MAROC.)

(116) National kidney foundation. About kidney transplant. Avaibale from http://www.nkfs.org/treatment/kidney-transplant/about-kidney-transplant/

(117) Food and Agricultural Organization: World Health Organization: United Nations University Expert consultation. Report on humanenergyrequirements. Rome; FAO, 2004.

(118) British Nutrition foundation. Nutrition requirements revised nov 2015.

(119) http://sciencelearn.org.nz/Contexts/Digestion-Chemistry/Science-Ideas-and-Concepts/Energy-requirements-of-the-body

(120) The Department of Nutrition for Health and Development, in collaboration with FAO, Dietary recommendations / Nutritional requirements, avaibale from http://www.who.int/nutrition/topics/nutrecomm/en/

(121) British nutrition Foundation. Dietary calcium and health, avaibale from https://www.nutrition.org.uk/attachments/205_Dietary%20calcium%20and%20health%20summary.pdf

(122) Reports of the Scientific CornmitteelorFwd: 31sl series. chapter 19 Calcium.

(123) Leitch I. Aitken FC. (1959). The estimation of calcium requirement: a reexamination. NurrAhrtRru 29. '40%-dl 1

(124) KDOQI Clinical Practice Guidelines for Chronic Kidney Disease: Evaluation, Classification, and Stratification. PART 6. ASSOCIATION OF LEVEL OF GFR WITH COMPLICATIONS IN ADULTS .GUIDELINE 9. ASSOCIATION OF LEVEL OF GFR WITH NUTRITIONAL STATUS

(125) ESPEN Guidelines on Enteral Nutrition: Adult Renal Failure$ N. Canoa,, E. Fiaccadorib , P. Tesinskyc , G. Toigod , W. Drumle , DGEM:$$ M. Kuhlmann, H. Mann, W.H. Ho¨rl

(126) Cano N, Fiaccadori E, Tesinsky P et al.: ESPEN Guidelines on Enteral Nutrition: AdultRenalFailure. Clin Nutr 2006; 25: 295–310.

(127) Fouque D, Vennegoor M, ter Wee P et al.: EBPG Guideline on Nutrition. Nephrol Dial Transplant 2007; 22 (Suppl 2): ii45–ii87 http://ndt.oxfordjournals.org/cgi/content/full/22/suppl_2/ii45 (last visited 20.04.2010)

(128) NKF: KDOQI Clinical Practice Guidelines and Clinical Practice Recommendations for 2006 Updates: Haemodialysis Adequacy, Peritoneal Dialysis Adequacy and Vascular Access. Am J Kidney Dis 2006; 48 (Suppl 1): S1-S322. http://www.kidney.org/professionals/kdoqi/guidelines_commentaries.cfm(last visited 20.04.2010)

(129) Pasticci F., Fantuzzi A.L., Pegoraro M., McCann M., Bedogni G. (2012). Nutritional management of stage 5 chronic kidney disease. Journal of Renal Care38(1), 50-58

(130) Adaptedfrom: NKF 2000 (DOQI); Toigo et al.(2000) (ESPEN); EDTNA/ERCA 2002; Fouque et al.(2007) (EPBG)

(131) DOQI = Disease Outcomes Quality Initiative; ESPEN = European Society for Parenteral and Enteral Nutrition; EBPG = European Best Practice Guidelines; EDTNA/ERCA = European Dialysis and Transplant Nurses Association/European

Renal Care Association;N/A = Not available. Adaptedfrom: NKF 2000 (DOQI); Toigo et al.(2000) (ESPEN); EDTNA/ERCA 2002; Fouque et al. (2007)(EPBG).

(132) Fouque D, Vennegoor M, ter Wee P et al.: EBPG Guideline on Nutrition. Nephrol Dial Transplant 2007; 22 (Suppl 2): ii45–ii87http://ndt.oxfordjournals.org/cgi/content/full/22/suppl_2/ii45 (last visited 20.04.2010)

(133) NKF-K/DOQI CKD Guidelines, GUIDELINE 5: NUTRITIONAL MANAGEMENT IN DIABETES AND CHRONIC KIDNEY DISEASE

(134) K/DOQI Clinical Practice Guidelines on Hypertension and Antihypertensive Agents in Chronic Kidney Disease. GUIDELINE 6: DIETARY AND OTHER THERAPEUTIC LIFESTYLE CHANGES IN ADULTS

(135) American Journal of Kidney Diseases (AJKD): KDOQI Clinical Practice Guideline for Nutrition in Children with CKD: 2008 Update

(136) Foreman JW, AbitbolCL,Trachtman H, et al: Nutritional intake in children with renal insufficiency: A report of The growth failure in children with renal diseases study.Jam CollNutr15:579-585,1996

(137) CanepaA,Perfumo F, Carrea A, etal: Protein and calorie intake, nitrogen losses, and nitrogen balance in children undergoing chronic peritoneal dialysis. AdvPerit Dial12:326-329,1996

(138) EdefontiA,Picca M, DamianiB,et al: Dietary prescription based on estimated nitrogen balance during peritoneal dialysis. PediatrNephrol 13:253-258,1999

(139) Arnold WC, Danford D, Holliday MA. Effects of caloric supplementation on growth in children with uremia. Kidney Int 1983;24:205–209.

(140) Norman LJ,Coleman JE, MacdonaldI A, Tomsett AM, Watson AR: Nutrition and growth in relation to severity Of renal disease in children. PediatrNephrol 15:259-265,2000

(141) OrejasG,Santos F, Malaga S,ReyC,Cobo A, Simarro M: Nutritional status of children with moderate chronic renal failure.Pediatr Nephrol9:52-56,1995

(142) Holliday MA. Caloric deficiency in children with uremia: effect upon growth. Pediatrics 1972;50:590–597.

(143) The National Kidney and Urologic Diseases Information Clearinghouse (NKUDIC): Nutrition in children with chronic kidney disease.

(144) DOQI Clinical Practice Guidelines for Nutrition in Chronic Renal Failure: pediatric guidelines: Energy intake for children with maintenance dialysis.

(145) KDOQI Clinical Practice Guideline for Nutrition in Children with CKD: 2008 Update. RECOMMENDATION 8: FLUID AND ELECTROLYTE REQUIREMENTS AND THERAPY

(146) National Kidney Foundation:K/DOQI clinical practice guidelines on hypertension and anti hypertensive agents in chronic kidney disease.AmJ Kidney Dis43:S1-S290,2004

(147) royer M, Folio D, MosserF . Diététique et néphropathie de l'enfant. EMC-Pédiatrie 1(2004) 281-295

(148) Evidence based practice guidelines for the nutritional management of adult kidney transplant recipients, developed by THE NEW SOUTH WALES RENAL SERVICES NETWORK TRANSPLANT WORKING GROUP, Greater Metropolitan Clinical Taskforce 2009.

(149) L'evaluation de l'etat nutritionnel chez le dialysé chronique; thesepresentée et soutenue publiquement le 15/03/2011 par meryemEssabani, au sein de la faculté de médecine et de pharmacie de Fes.

(150) Cogan MG, Sargent JA, Yarborough SG, Vincenti F, Amend Jr WJ. Prevention of prednisone-induced negative nitrogen balance. Annals of internal Medicine 1981;95;158-61.

(151) Whittier FC, Evans DH, Dutton S, et al. Nutrition in renal transplantation. AM J Kidney Dis 1985;6:405-11.

(152) National Health and Medical research Council. Nutrient reference values for Australia and New Zealand. Canberra: NHMRC; 2005.

(153) National Heart Foundation of Australia (National Blood pressure and vascular disease Advisory Committee). Guide to management of hypertension 2008. Assessing and managing raised blood pressure in adults. 2008

(154) National Heart Foundation of Australia. Hypertension management guide for doctors. 2004

(155) Palmer SC, McGregor DO, Strippoli GFM. Interventions for preventing bone disease in kidney transplant recipients. Cochrane Database of systematic reviews issue 32007.

(156) KOC M, Tuglular H, Arikan H, Ozener C, E, A. Alendronate increases bone mineral density in long-term renal transplant recipients. Transplant proc2002 ;34: 21 22-3.

(157) The freedictionary (on-line). Available from : http://medical-disctionary.thefreedictionary.com

(158) Stenvinkel P, heimburger O, Lindholm B, kaysen GA, bergstrom J. Are there two types of malnutrition in chronic renal failure? Evidence for relationships between malnutrition, inflammation and atherosclerosis (MIA syndrome). Nephrol Dial Transplant 15(7):953-690,2000.

(159) Kopple J: Effect of nutrition on morbidity and mortality in maintenance dialysis patients. Am J Kidney Dis 24:1002-9, 1994

(160) Mehrotra R and Kopple JD: Nutritional Management of Maintenance Dialysis Patients: Why Aren't We Doing Better? Ann Rev Nutr 21:343-379, 2001

(161) Tchekmedyian N, Zahyna D, Halpert C, and Heber D: Assessment and maintenance of nutrition in older cancer patients. Oncology (Huntingt) 6:105-11, 1992

(162) Cederholm T, Jagren C, and Hellstrom K: Outcome of protein-energy malnutrition in elderly medical patients. Am J Med 98:67-74, 1995

(163) Nutritional factors and clinical outcomes of patients with end stage kidney disease: implications for practice from researching the practice framework Maria Chi-Fei Chan University of Wollongong

(164) Nutritional management in pre-dialysis, chronic kidney disease, chronic kidney disease. These is submitted by Katrina Louis campbele. In the institute of health and biomedical innovation, school of public health queensland university of technology. 2007.

(165) Carrero JJ. Identification of patients with eating disorders: clinical and biochemical signs of appetite loss in dialysis patients. J Ren Nutr. 2009;19(1):10-5

(166) Bergstrom J. Mechanisms of uremic suppression of appetite. J Ren Nutr. 1999;9(3):129-32.

(167) Stenvinkel P, Heimbürger O, Lindholm B, Kaysen GA, Bergström J. Are there two types of malnutrition in chronic renal failure? Evidence for relationships between malnutrition, inflammation and atherosclerosis (MIA syndrome). Nephrol Dial Transplant. 2000;15(7):953-60.

(168) Stenvinkel P. Inflammatory and atherosclerotic interactions in the depleted uremic patient. Blood Purif. 2001;19(1):53-61.

(169) Kaysen GA, Rathore V, Shearer GC, Depner TA. Mechanisms of hypoalbuminemia in hemodialysis patients. Kidney Int. 1995;48(2):510-6.

(170) Struijk DG, Krediet RT, Koomen GC, Boeschoten EW, Arisz L. The effect of serum albumin at the start of continuous ambulatory peritoneal dialysis treatment on patient survival. Perit Dial Int. 1994;14(2):121-6.

(171) Honda H, Qureshi AR, Heimburger O, Barany P, Wang K, Pecoits-Filho R, et al. Serum albumin, C-reactive protein, interleukin 6, and fetuin a as predictors of malnutrition, cardiovascular disease, and mortality in patients with ESRD. Am J Kidney Dis. 2006;47(1):139-48.

(172) kizler TA. Nutrition, inflammation and chronic kidney disease. CurrOpinNephrolHypertens. 2008;17(2):162-7.

(173) Stenvinkel P, Barany P, Chung SH, Lindholm B, Heimburger O. A comparative analysis of nutritional parameters as predictors of outcome in male and female ESRD patients. Nephrol Dial Transplant. 2002;17(7):1266-74

(174) Kalantar-Zadeh K, Ikizler TA, Block G, Avram MM, Kopple JD. Malnutrition-inflammation complex syndrome in dialysis patients: causes and consequences. Am J Kidney Dis. 2003;42(5):864-81.

(175) Chen W, Abramowitz MK. Treatment of metabolic acidosis in patients with CKD. Am J Kidney Dis. 2014;63(2):311-7.

(176) Ballmer P, Imoberdorf R. Influence of acidosis on protein metabolism. Nutrition. 1995;11(5):462-8; discussion 70.

(177) Bommer J, Locatelli F, Satayathum S, Keen ML, Goodkin DA, Saito A, et al. Association of predialysis serum bicarbonate levels with risk of mortality and hospitalization in the Dialysis Outcomes and Practice Patterns Study (DOPPS). Am J Kidney Dis. 2004;44(4):661-71.

(178) Lowrie EG, Lew NL. Death risk in hemodialysis patients: the predictive value of commonly measured variables and an evaluation of death rate differences between facilities. Am J Kidney Dis. 1990;15(5):458-82

(179) Kovesdy CP, Anderson JE, Kalantar-Zadeh K. Association of serum bicarbonate levels with mortality in patients with non-dialysis-dependent CKD. Nephrol Dial Transplant. 2009;24(4):1232-7.

(180) Garibotto G, Sofia A, Saffioti S, Bonanni A, Mannucci I, Parodi EL, et al. Effects of peritoneal dialysis on protein metabolism. NutrMetabCardiovasc Dis. 2013;23 Suppl 1:S25-30

(181) Lim VS, Bier DM, Flanigan MJ, Sum-Ping ST. The effect of hemodialysis on protein metabolism. A leucine kinetic study. J Clin Invest. 1993;91(6):2429-36

(182) Ikizler TA. Effects of hemodialysis on protein metabolism. J Ren Nutr. 2005;15(1):39-43

(183) 18 Caglar K, Fedje L, Dimmit R. Therapeutic effects of oral nutritional supplementation during hemodialysis. Kidney Int 2002;62:1054-9.

(184) Cano N. Nutrition de l'hémodialysé chronique. NutrClin et Metab 2004;18:7-10.

(185) Pupim LB, Cuppari L, and Ikizler TA: Nutrition and Metabolism in Kidney Disease. SeminNephrol 26:134-157, 2006

(186) Denning PJ, Comer DE, Gries D, Mulder MC, Tucker A, Turner AJ, et al. Computing as a discipline. COMPUTER,. 1989;:63-70.

(187) National Kidney Foundation.GFR calculator formulas, CKD-EPI Creatinine equation (2009), MDRD study equation. Available on : https://www.kidney.org/professionals/kdoqi/gfr_calculator

(188) Oman Med J. 2012 Mar; 27(2): 108–113. doi: 10.5001/omj.2012.23. Estimated Glomerular Filtration Rate (eGFR): A Serum Creatinine-Based Test for the Detection of Chronic Kidney Disease and its Impact on Clinical Practice

(189) ClinBiochem Rev. 2011 May; 32(2): 75–79. Methods of Estimating GFR – Different Equations Including CKD-EPI

(190) Sanofi Nutridial application available from : http://www.wopata.com/blog/2015/7/sanofi-nutridial-app-ios-android-un-suivi-continu-patients-dialyses

(191) Sanofi renal KidneyAppetite application, available from: http://www.kidneyappetite.com/

(192) Site interactif pour enquete alimentaire http://www.bdinteractivemedia.com/Diet_Tool/

(193) Android studio website.

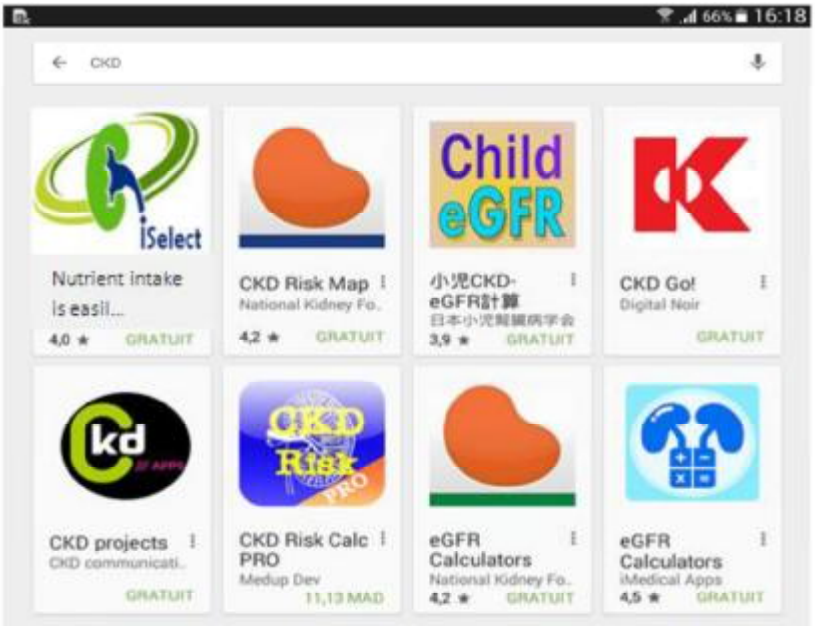
(194) Medical application in kidney disease, by the nationa kidney foundation. Available from :https://www.kidney.org/apps

APPENDICES

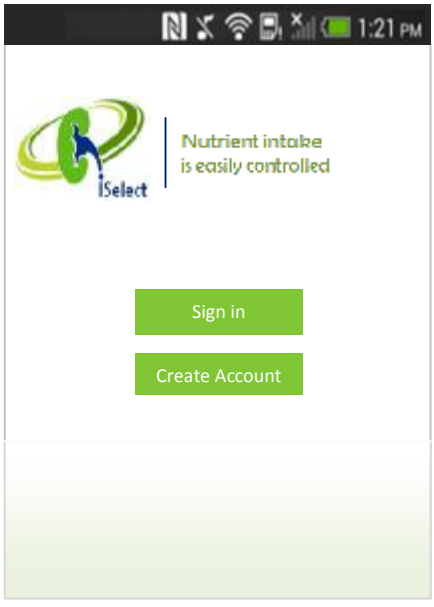
Design of iSelect application (English version)

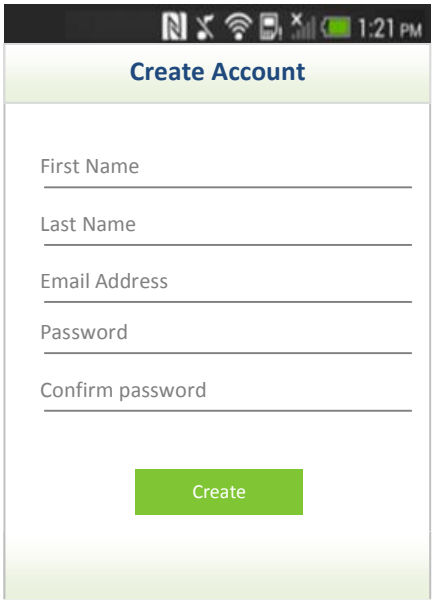
Application icôn/logo

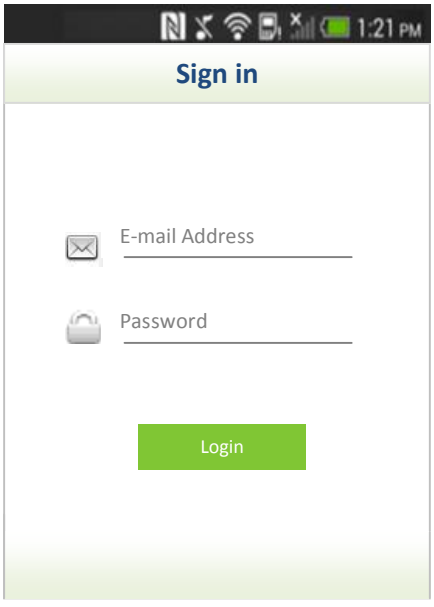
iSelect
Nutrient intake is easily controlled



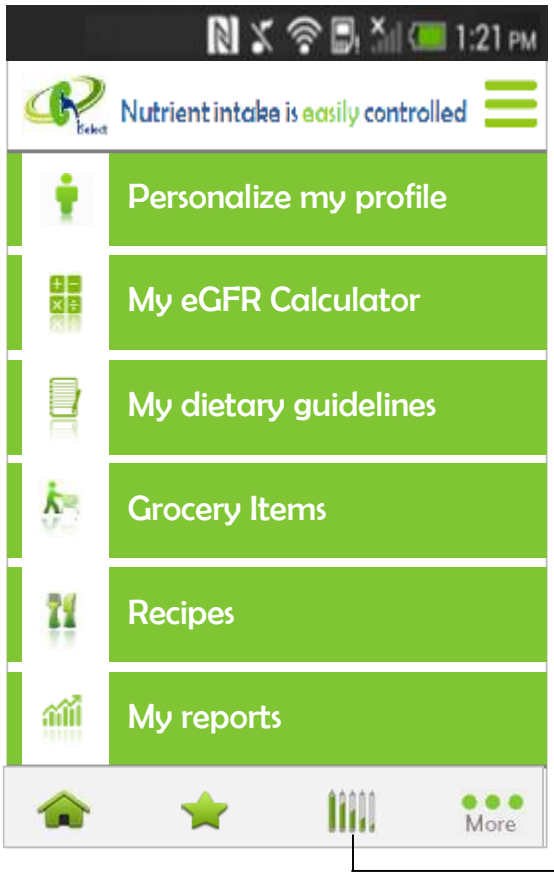
Authentication pages :

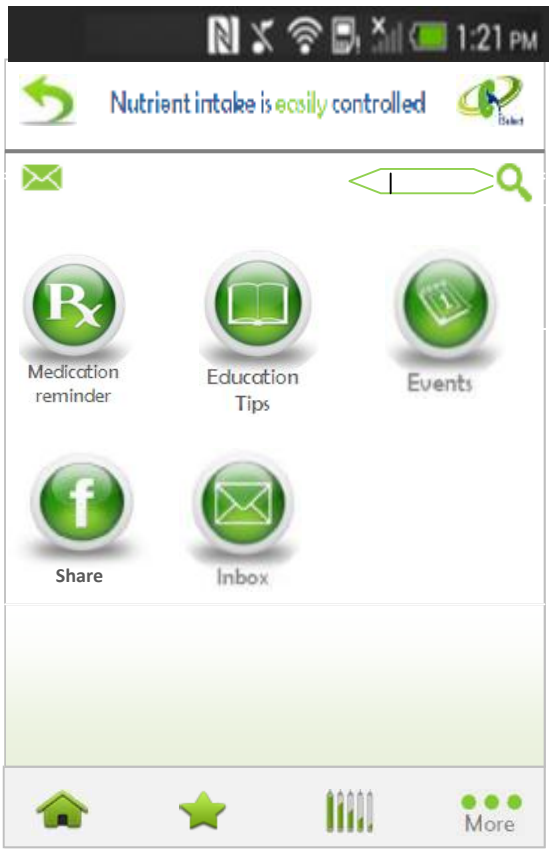


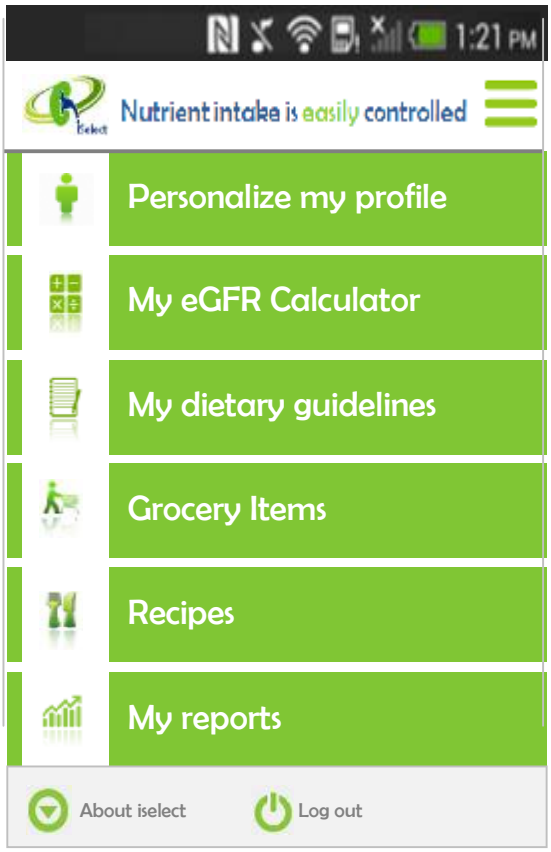




Features

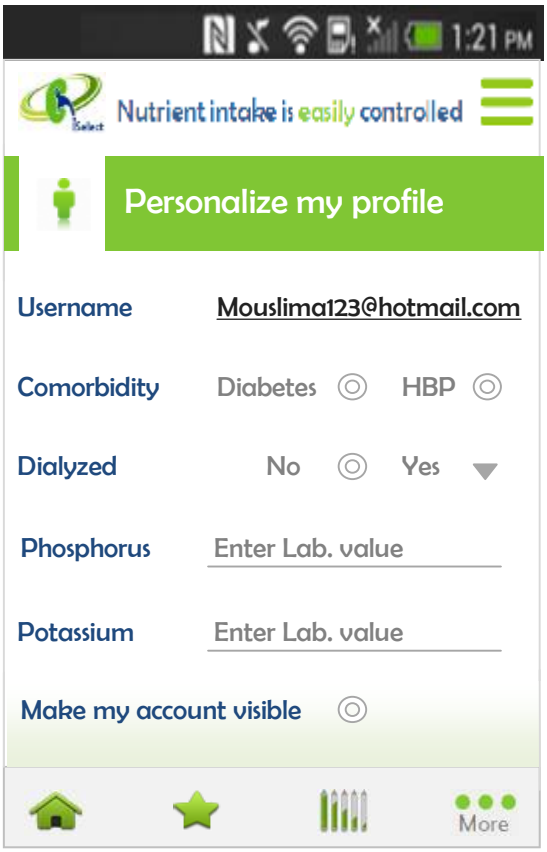

1:21 PM
Nutrient intake is easily controlled
Personalize my profile
My eGFR Calculator
My dietary guidelines
Grocery Items
Recipes
My reports
More

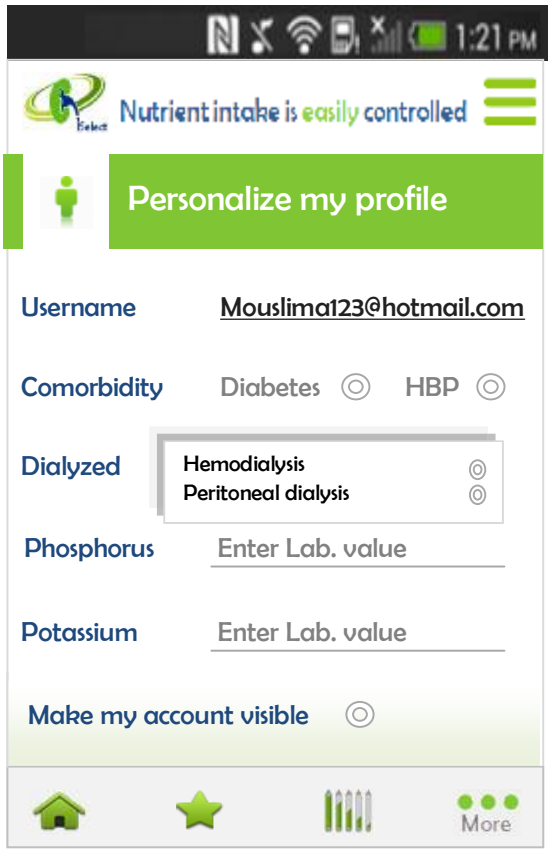

1:21 PM
Nutrient intake is easily controlled
Medication reminder
Education Tips
Events
Share
Inbox
More


1:21 PM
Nutrient intake is easily controlled
Personalize my profile
My eGFR Calculator
My dietary guidelines
Grocery Items
Recipes
My reports
About iselect
Log out

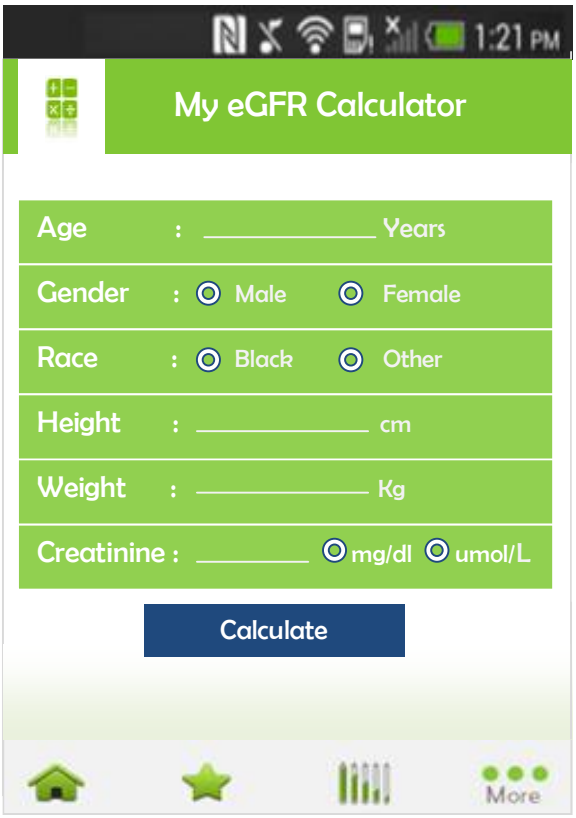

1:21 PM
Nutrient intake is easily controlled
Monday, April 29
32%
Phosphorus
98%
Potassium
5%
Sodium
72%
Calcium
125%
Fluid
10%
Protein
92%
Calories
16%
Carbohydrate
More

Personalize my profile

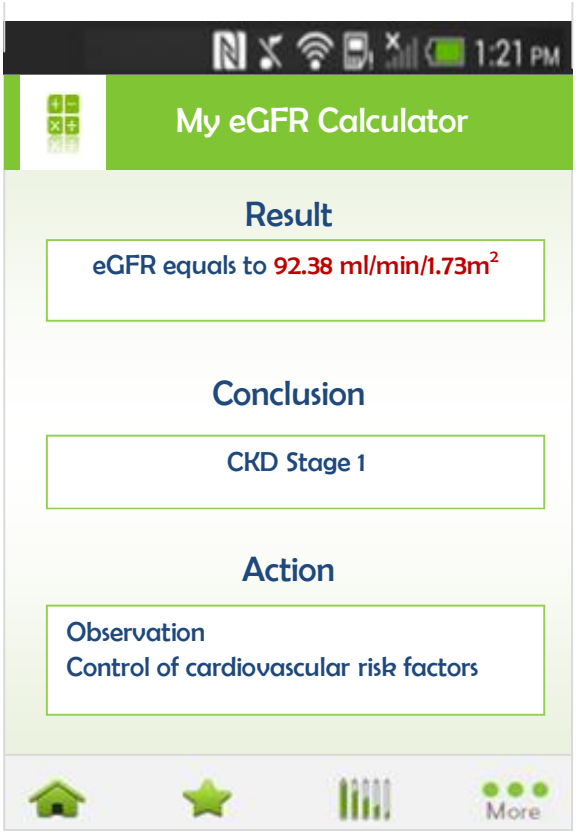




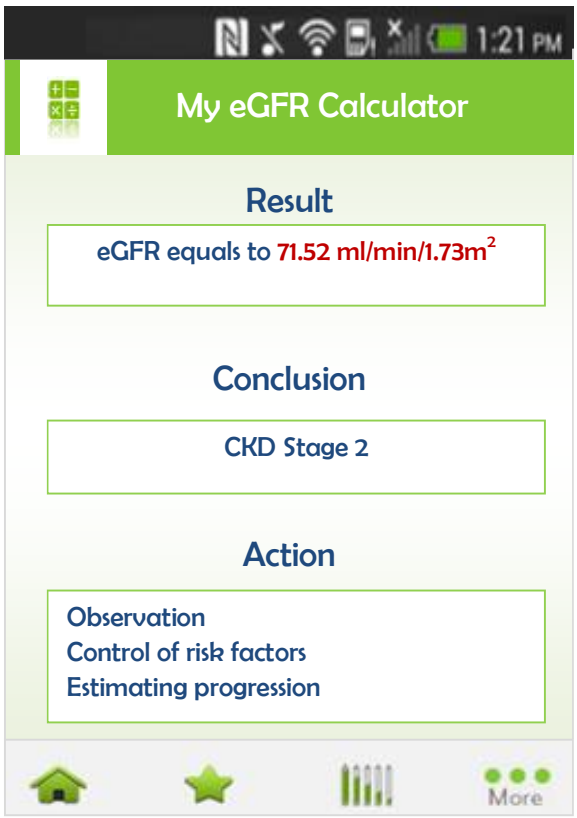
My eGFR calculator



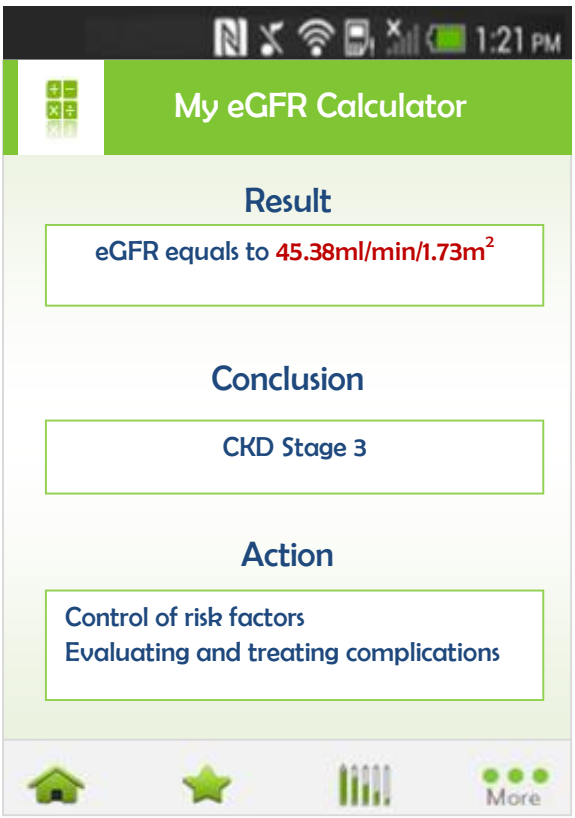
eGFR >= 90ml/min/1.73m^2



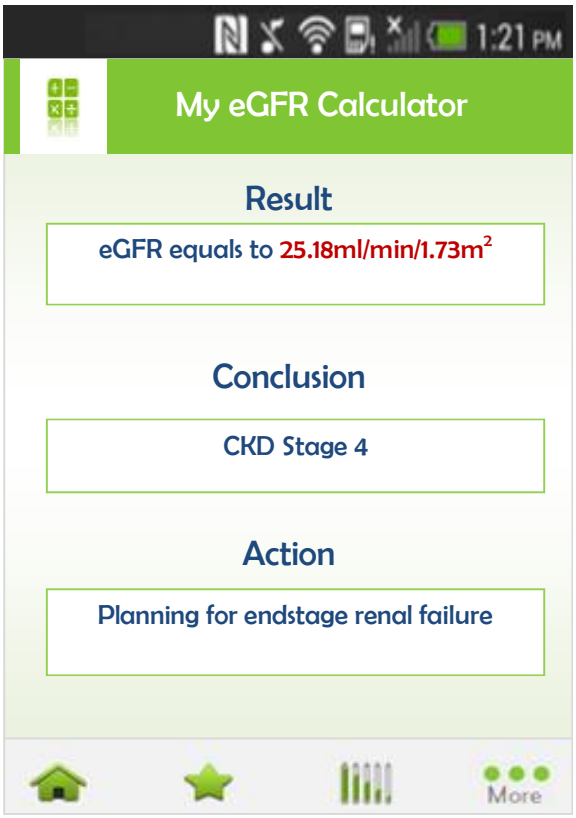
60 ≤ eGFR ≤ 89



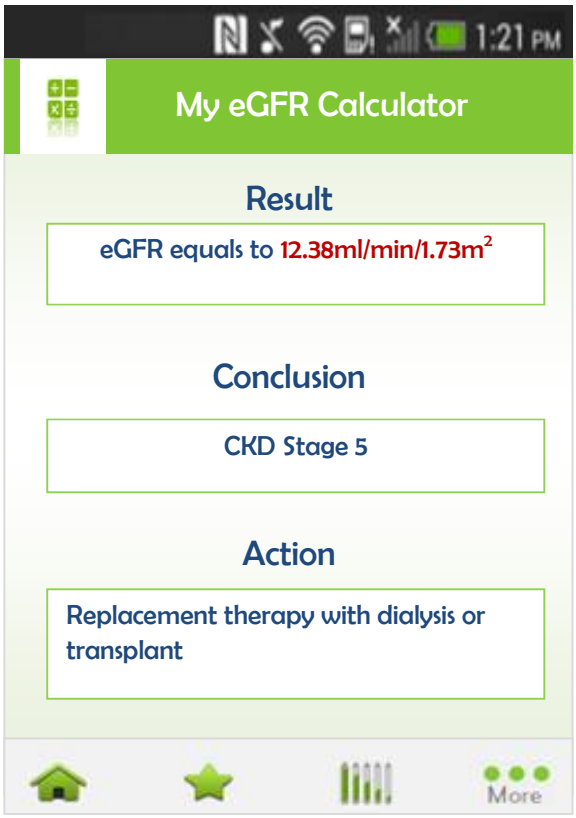
30 ≤ eGFR ≤ 59



15 ≤ eGFR ≤ 29



eGFR < 15



My dietary guidelines

Adults ≥ 18 years ; non diabetics, without HBP, kaliemie>5.5 mmol/l , phosphoremie>70mg/l

CKD Stage 1 (<60years)

CKD Stage 1 (age ≥ 60 years)

CKD Stage 2 (<60years)

CKD Stage 2 (age ≥ 60 years)

CKD Stage 3 (<60years)

CKD Stage 3 (age ≥ 60 years)

Adults ≥ 18 years ; non diabetics, without HBP, kaliemie>5.5 mmol/l , phosphoremie < 70mg/l

CKD Stage 1 (<60years)

CKD Stage 1 (age ≥ 60 years)

CKD Stage 2 (<60years)

CKD Stage 2 (age ≥ 60 years)

CKD Stage 3 (<60years)

CKD Stage 3 (age ≥ 60 years)

25 < eGFR ≤ 29

CKD Stage 4 (<60years)

CKD Stage 4 (age ≥ 60 years)

15 < eGFR ≤ 25

CKD Stage 4 (<60years)

CKD Stage 4 (age ≥ 60 years)

CKD stage 5 (Non dialyzed)

CKD Stage 5 (<60years)

CKD Stage 5 (age ≥ 60 years)

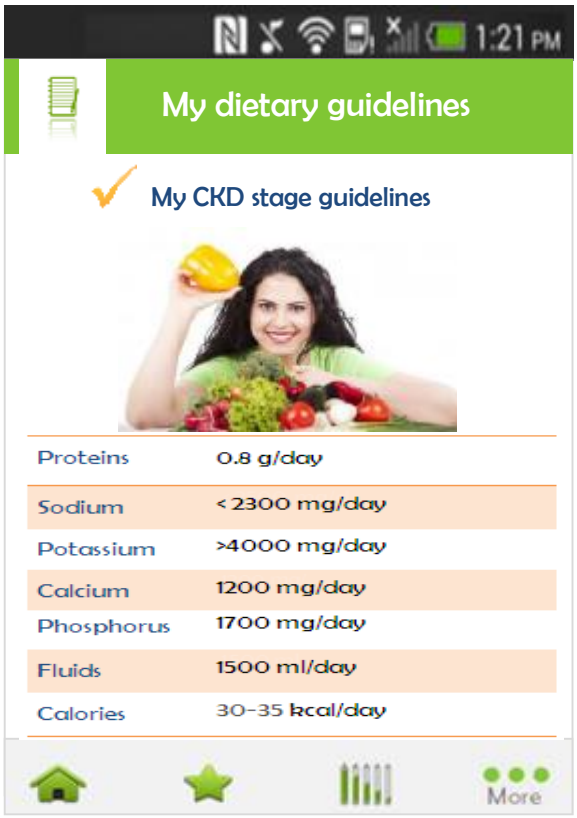
CKD stage 5 (dialyzed)

Hemodialysis

Peritoneal dialysis

Adults ≥ 18 years ; diabetics, without HBP

Diabeties CKD Stage 1- 2



Diabeties CKD Stage 3- 4

Diabeties CKD Stage 5(non dialyzed)

Diabeties Stage 5 (Hemodialysis)

Diabeties Stage 5 (Peritoneal dialysis)

Adults ≥ 18 years ; non diabetics, with HBP, kaliemie >5.5 mmol/l, phosphoremie > 70 mg/l

HBP CKD Stage 1 (<60years)

HBP CKD Stage 1 (age ≥ 60 years)

HBP CKD Stage 2 (<60years)

HBP CKD Stage 2 (age ≥ 60 years)

HBP CKD Stage 3 (<60years)

HBP CKD Stage 3 (age ≥ 60

Adults ≥ 18 years ; non diabetics, with HBP, kaliemie < 5.5 mmol/l, phosphoremie <70 mg/l

HBP CKD Stage 1 (<60years)

HBP CKD Stage 1 (age ≥ 60 years)

HBP CKD Stage 2 (<60years)

HBP CKD Stage 2 (age ≥ 60 years)

HBP CKD Stage 3 (<60years)

HBP CKD Stage 3 (age ≥ 60

25 < eGFR ≤ 29

HBP CKD Stage 4 (<60years)

HBP CKD Stage 4 (age ≥ 60

15 < eGFR ≤ 25

HBP CKD Stage 4 (<60years)

HBP CKD Stage 4 (age ≥ 60 years)

CKD stage 5 (Non dialyzed)

HBP CKD Stage 5 (<60years)

HBP CKD Stage 5 (age ≥ 60

CKD stage 5 (dialyzed)

HBP Hemodialysis

HBP Peritoneal dialysis

Adults ≥ 18 years ; diabetics, with HBP

Diab+HBP CKD Stage 1- 2

1:21 PM

My dietary guidelines

My CKD stage guidelines

Proteins	0.8 g/day
Sodium	400-1000 mg/day
Potassium	>4000mg/day
Calcium	1200 mg/day
Phosphorus	1700 mg/day
Fluids	1500 ml/day
Calories	30-35 kcal/day

More

Diab+HBP CKD Stage 3- 4

1:21 PM

My dietary guidelines

My CKD stage guidelines

Proteins	0.6-0.8 g/day
Sodium	400-1000 mg/day
Potassium	2400mg/day
Calcium	1200 mg/day
Phosphorus	800-1000 mg/day
Fluids	1500 ml/day
Calories	30-35 kcal/day

More

Diab+HBP CKD Stage 5(non dialyzed)

1:21 PM

My dietary guidelines

My CKD stage guidelines

Proteins	1.3-1.5 g/day
Sodium	400-1000 mg/day
Potassium	2000-3000 mg/day
Calcium	<1500 mg/day
Phosphorus	800-1000 mg/day
Fluids	1500 ml/day
Calories	35-40 kcal/day

More

Diab+HBP Stage 5 (Hemodialysis)

1:21 PM

My dietary guidelines

My CKD stage guidelines

Proteins	1.2-1.4 g/day
Sodium	400-1000 mg/day
Potassium	2000-2500 mg/day
Calcium	<2000 mg/day
Phosphorus	800-1000 mg/day
Fluids	500-1000 ml/day
Calories	35-40 kcal/day

More

Diab+HBP Stage 5 (Peritoneal dialysis)

Children (≤ 18 years)

0-6months (pre-dialysis normal phosphorus)

0-6months (pre-dialysis high phosphorus)

0-6months (hemodialysis, normal phosphorus)

0-6months (hemodialysis, high phosphorus)

0-6months (peritoneal dialysis, normal phosphorus)

0-6months (peritoneal dialysis, high phosphorus)

7-12 months (pre-dialysis, normal phosphorus)

7-12 months (pre-dialysis, high phosphorus)

7-12 months (hemodialysis, normal phosphorus)

7-12 months (hemodialysis, high phosphorus)

7-12 months (peritoneal dialysis, normal phosphorus)

7-12 months (peritoneal dialysis, high phosphorus)

1 to 3 years : Girls (see boys in the other document)

1-3 years (pre-dialysis; normal phosphorus)

1-3 years (pre-dialysis; high phosphorus)

1-3 years (hemodialysis; normal phosphorus)

1-3 years (hemodialysis; high phosphorus)

1-3 years (peritoneal dialysis; normal phosphorus)

1-3 years (peritoneal dialysis; high phosphorus)

4 to 6 years : Girls (see boys in the other document)

4-6 years (pre-dialysis, normal phosphorus)

4-6 years (pre-dialysis, high phosphorus)

4-6 years (hemodialysis, normal phosphorus)

4-6 years (hemodialysis, high phosphorus)

4-6 years (peritoneal dialysis, normal phosphorus)

4-6 years (peritoneal dialysis, high phosphorus)

7 to 10 years : Girls (see boys in the other document)

7-10 years (predialysis, normal phosphorus)

7-10 years (predialysis, high phosphorus)

7-10 years (hemodialysis, normal phosphorus)

7-10 years (hemodialysis, high phosphorus)

7-10 years (peritoneal dialysis, normal phosphorus)

7-10 years (peritoneal dialysis, high phosphorus)

GIRLS

11-14 years (predialysis, normal phosphorus)

11-14 years (predialysis, high phosphorus)

11-14 years (hemodialysis, normal phosphorus)

11-14 years (hemodialysis, high phosphorus)

11-14 years (peritoneal dialysis, normal phosphorus)

11-14 years (peritoneal dialysis, high phosphorus)

Boys

11-14 years (predialysis, normal phosphorus)

11-14 years (predialysis, high phosphorus)

11-14 years (hemodialysis, normal phosphorus)

11-14 years (hemodialysis, high phosphorus)

11-14 years (peritoneal dialysis, normal phosphorus)

11-14 years (peritoneal dialysis, high phosphorus)

Girls

15-18 years (predialysis, normal phosphorus)

15-18 years (predialysis, high phosphorus)

15-18 years (hemodialysis, normal phosphorus)

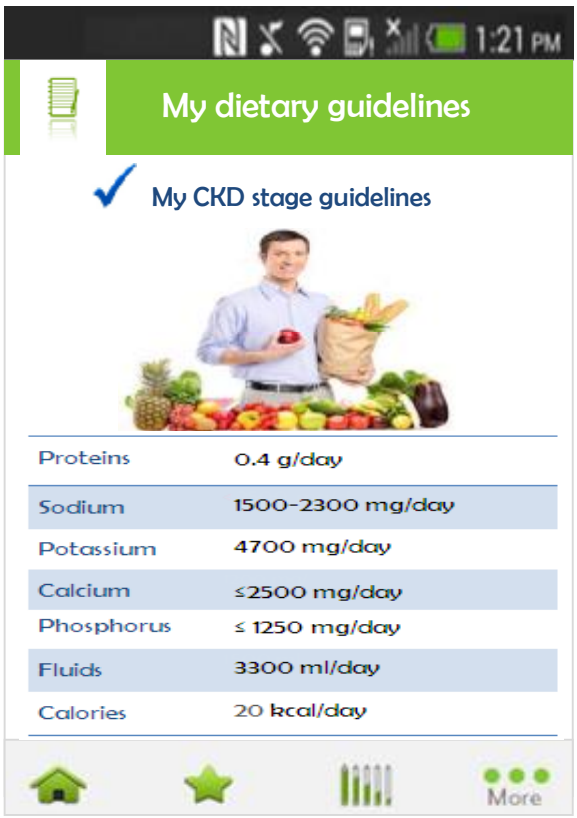
15-18 years (hemodialysis, high phosphorus)

15-18 years (peritoneal dialysis, normal phosphorus)

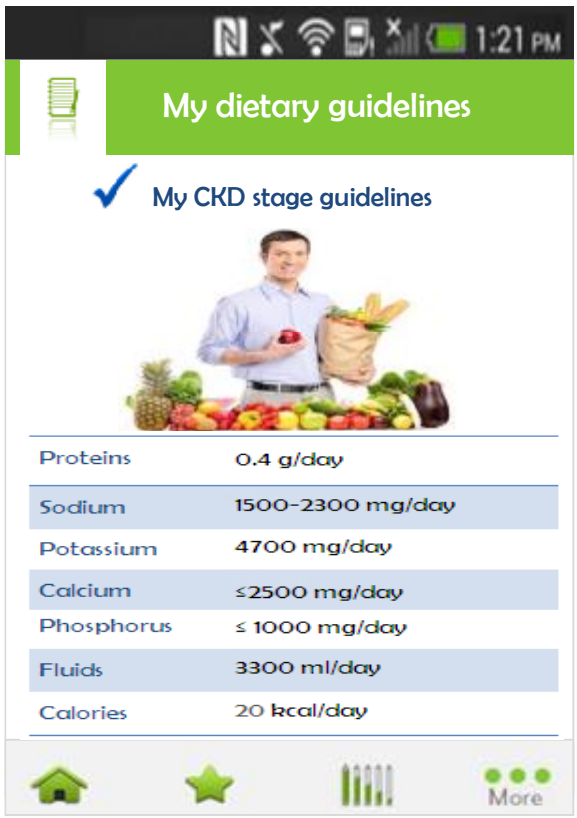
15-18 years (peritoneal dialysis, high phosphorus)

Boys

15-18 years (predialysis, normal phosphorus)



15-18 years (predialysis, high phosphorus)



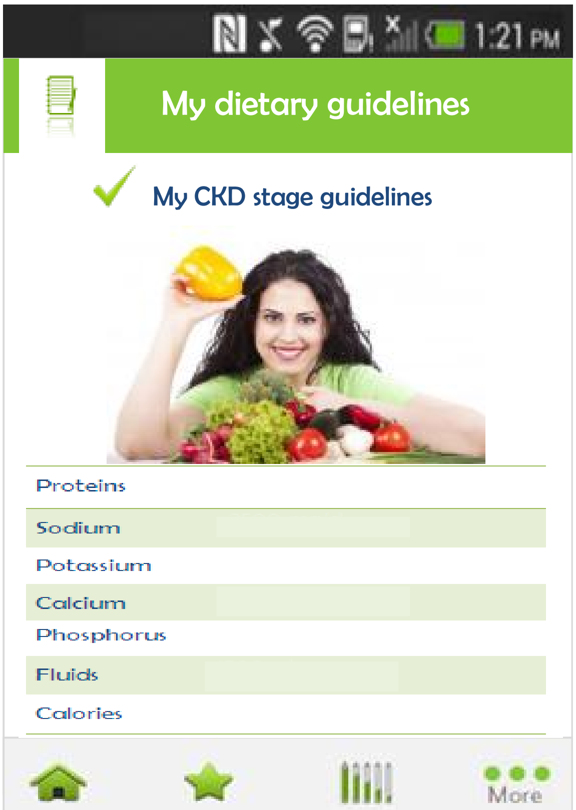
15-18 years (hemodialysis, normal phosphorus)

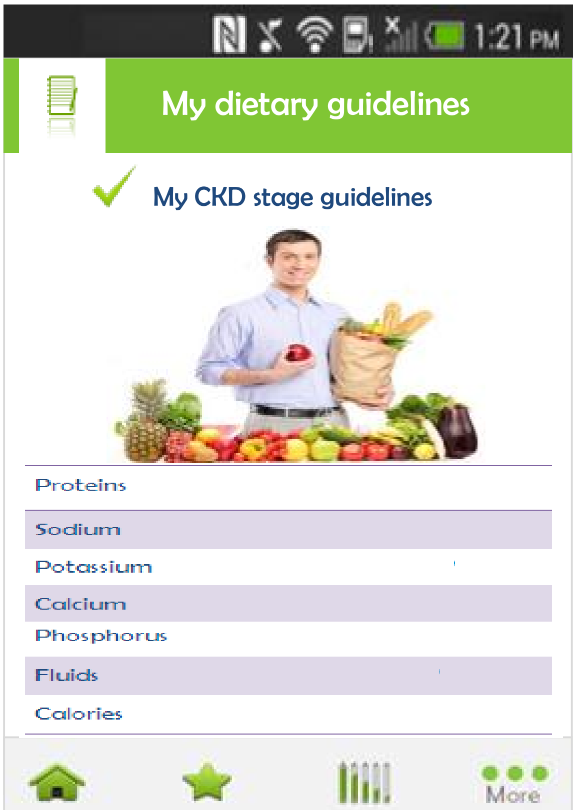
15-18 years (hemodialysis, high phosphorus)

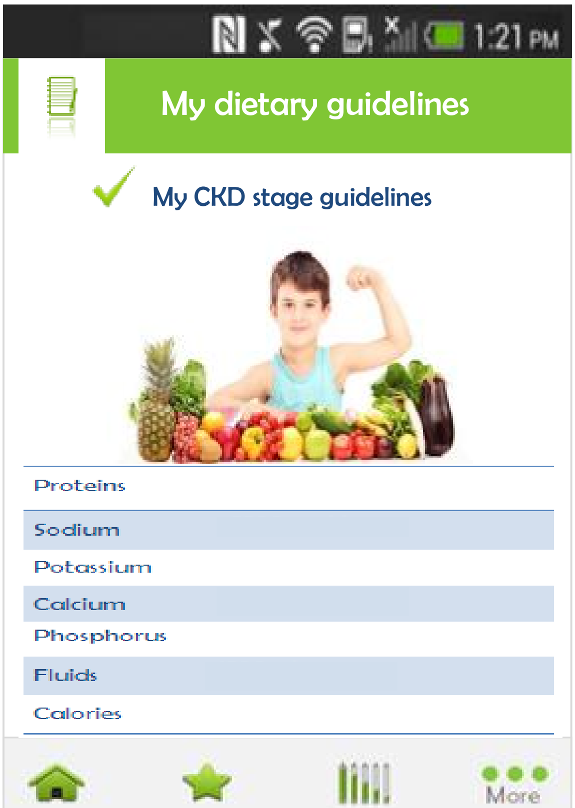
15-18 years (peritoneal dialysis, normal phosphorus)

15-18 years (peritoneal dialysis, high phosphorus)

Left for the Doctor recommendations

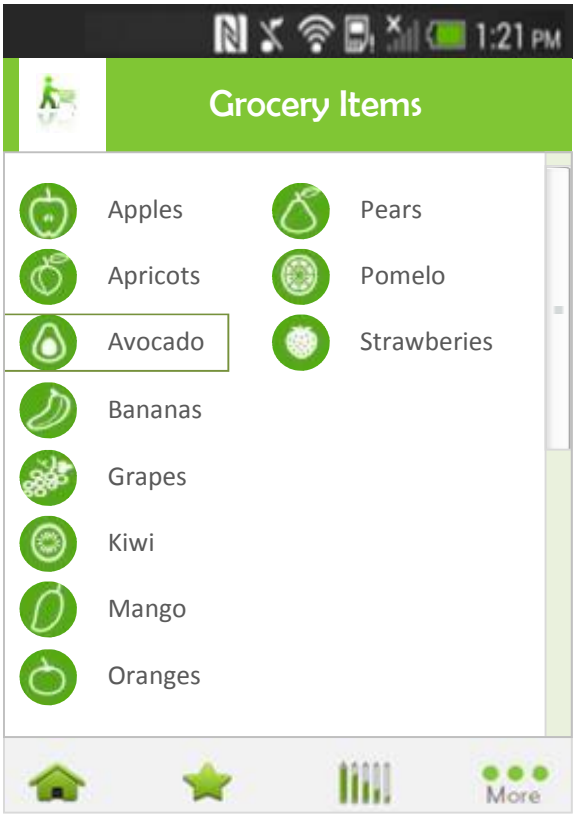


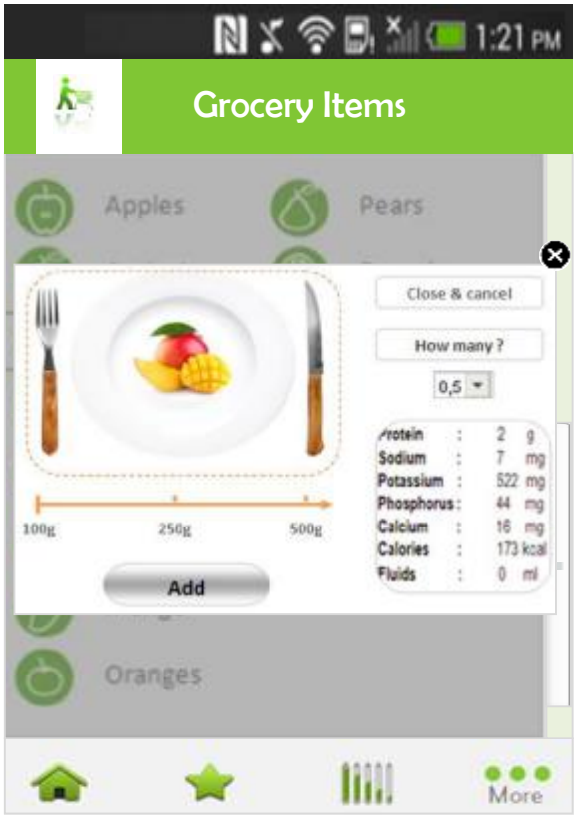


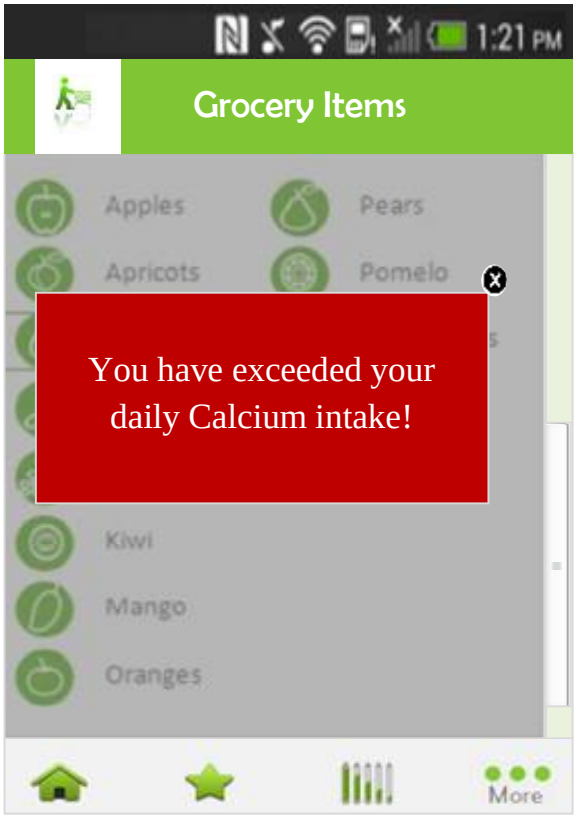



1:21 PM
My dietary guidelines
My CKD stage guidelines
Proteins
Sodium
Potassium
Calcium
Phosphorus
Fluids
Calories
More

Grocery items




1:21 PM
Grocery Items
Apples
Pears
Close & cancel
How many ?
0,5
Protein : 2 g
Sodium : 7 mg
Potassium : 522 mg
Phosphorus : 44 mg
Calcium : 16 mg
Calories : 173 kcal
Fluids : 0 ml
100g
250g
500g
Add
Oranges
More


1:21 PM
Grocery Items
Apples
Pears
Apricots
Pomelo
You have exceeded your daily Calcium intake!
Kiwi
Mango
Oranges
More

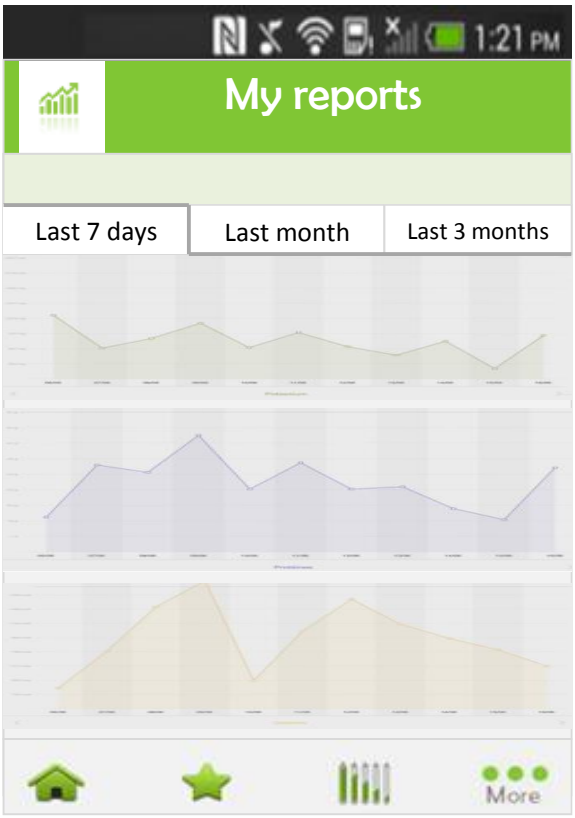
Recipes


1:21 PM
Recipes
Couscous with veal
Lentils
Rfissa with chicken
Pastilla with chicken
Chicken baldi
Recipe
Composition
More

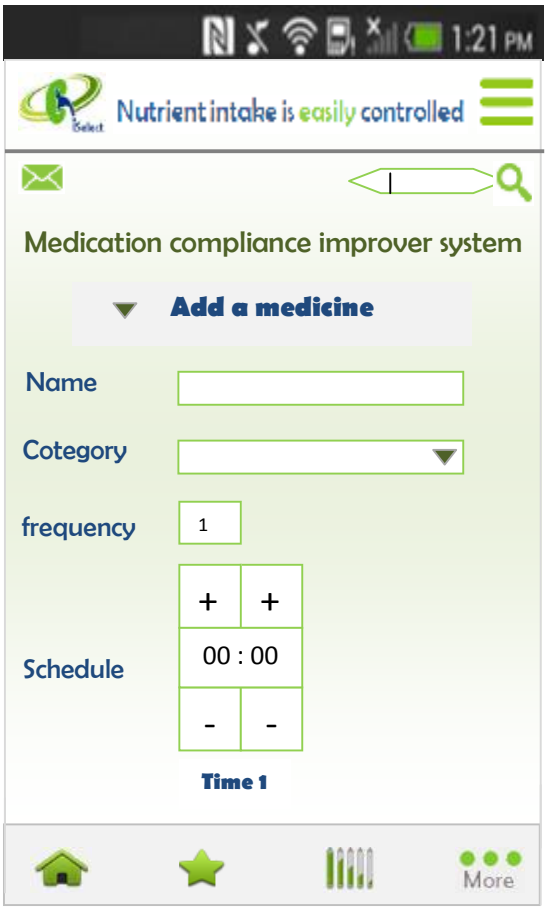

1:21 PM
Recipes
Hargma (calf's foot with chick-peas)
Ingredient: 6 to 8 persons
1 calf's foot cut into pieces
250g chick-peas (soaked overnight)
1 big onion
10 garlic cloves
Salt
½ teaspoon pepper
½ teaspoon ginger
Some saffron
½ teaspoon cinnamon,
½ teaspoon paprika
1 bunch parsley and coriander
Water
A white and clean cloth
250g wheat seeds
More

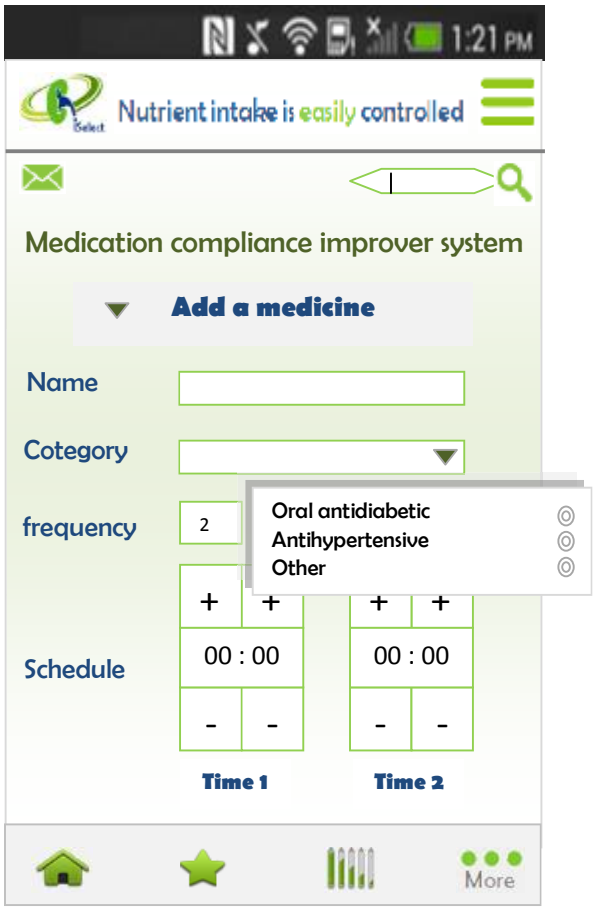

1:21 PM
Recipes
More

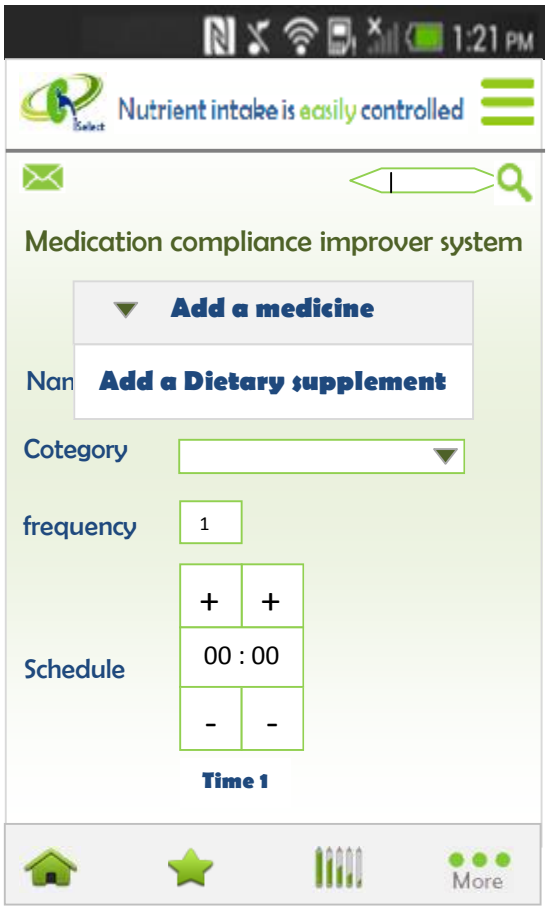
My reports

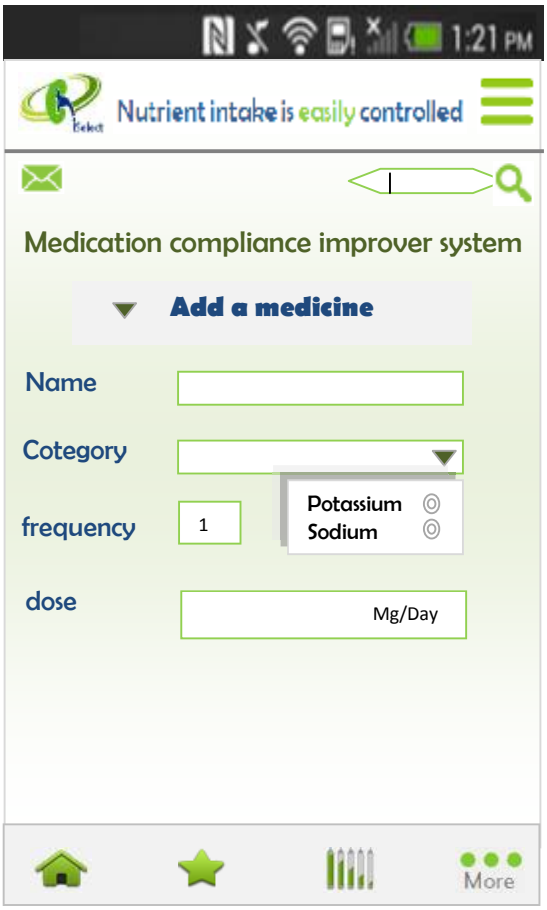


MCIS


1:21 PM
Nutrient intake is easily controlled
Medication compliance improver system
Add a medicine
Name
Category
frequency
1
Schedule
+
+
00 : 00
-
-
Time 1
More


1:21 PM
Nutrient intake is easily controlled
Medication compliance improver system
Add a medicine
Name
Category
frequency
2
Oral antidiabetic
Antihypertensive
Other
Schedule
00 : 00
00 : 00
Time 1
Time 2
More


1:21 PM
Nutrient intake is easily controlled
Medication compliance improver system
Add a medicine
Nan
Add a Dietary supplement
Cotegory
frequency
1
Schedule
+
+
00 : 00
-
-
Time 1
More

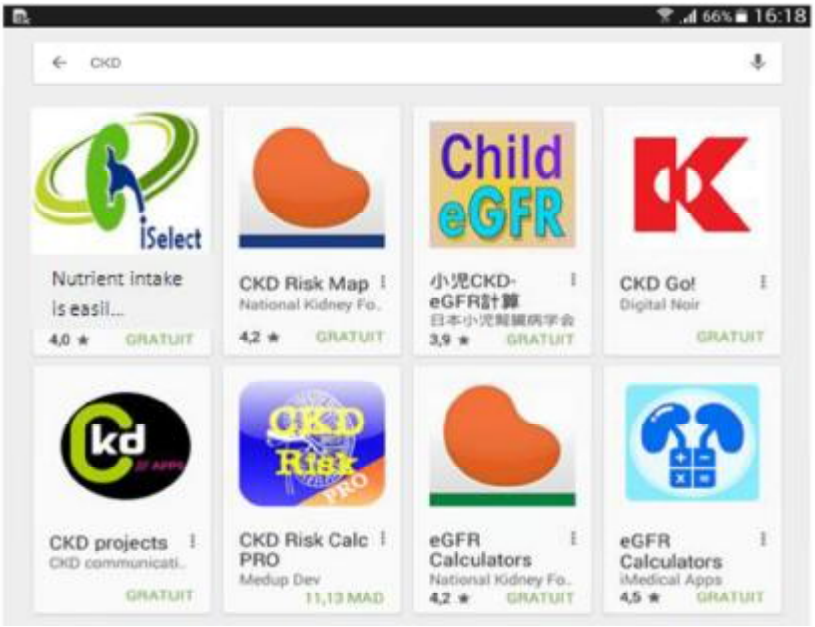

1:21 PM
Nutrient intake is easily controlled
Medication compliance improver system
Add a medicine
Name
Cotegory
frequency
1
Potassium
Sodium
dose
Mg/Day
More

تصميم التطبيق iSelect

النسخة العربية

شعار و أيقونة التطبيق

iSelect
Nutrient intake is easily controlled



صفحات التسجيل

الصفحات الرئيسية

تعديل ملفي الشخصي

حساب معدل الترشيح الكبيبي

eGFR>= 90ml/min/1.73m^2

60 ≤eGFR≤89

30 ≤eGFR≤59

15 ≤eGFR≤29

eGFR< 15

المبادئ التوجيهية الغذائية

Adults ≥ 18 years ; non diabetics, without HBP, kaliemie>5.5 mmol/l , phosphoremie>70mg/l

CKD Stage 1 (<60years)

CKD Stage 1 (age≥ 60 years)

CKD Stage 2 (<60years)

CKD Stage 2 (age≥ 60 years)

CKD Stage 3 (<60years)

CKD Stage 3 (age≥ 60 years)

Adults ≥ 18 years ; non diabetics, without HBP, kaliemie<5.5 mmol/l , phosphoremie< 70mg/l

CKD Stage 1 (<60years)

CKD Stage 1 (age≥ 60 years)

CKD Stage 2 (<60years)

CKD Stage 2 (age≥ 60 years)

CKD Stage 3 (age≥ 60 years)

CKD Stage 3 (<60years)

25 <eGFR≤ 29

CKD Stage 4 (<60years)

البروتينات	0.7 – 0.8 غ في اليوم
الصوديوم	<2500مغ في اليوم
البوتاسيوم	1500 – 1600مغ في اليوم
الكلسيوم	1200 مغ في اليوم
الفوسفور	600 – 1000مغ في اليوم
السوائل	1500 مل في اليوم
السعرات الحرارية	35≥ك كلر في اليوم

CKD Stage 4 (age≥ 60 years)

البروتينات	0.7 – 0.8غ في اليوم
الصوديوم	<2500مغ في اليوم
البوتاسيوم	1500 – 1600مغ في اليوم
الكلسيوم	1200مغ في اليوم
الفوسفور	600 – 1000مغ في اليوم
السوائل	1500 مل في اليوم
السعرات الحرارية	30-35ك كلر في اليوم

15<eGFR≤ 25

CKD Stage 4 (<60years)

البروتينات	0.6 غ في اليوم
الصوديوم	<2500 مغ في اليوم
البوتاسيوم	1500 – 1600مغ في اليوم
الكلسيوم	1200 مغ في اليوم
الفوسفور	600 – 1000مغ في اليوم
السوائل	1500 مل في اليوم
السعرات الحرارية	35≥ ك كلر في اليوم

CKD Stage 4 (age≥ 60 years)

البروتينات	0.6 غ في اليوم
الصوديوم	<2500مغ في اليوم
البوتاسيوم	1500 – 1600مغ في اليوم
الكلسيوم	1200 مغ في اليوم
الفوسفور	600 – 1000 مغ في اليوم
السوائل	1500 مل في اليوم
السعرات الحرارية	30-35 ك كلر في اليوم

CKD stage 5 (Non dialyzed)

CKD Stage 5 (<60years)

CKD Stage 5 (age≥ 60 years)

CKD stage 5 (dialyzed)

Hemodialysis

Peritonealdialysis

Adults ≥ 18 years ; diabetics, without HBP

DiabetiesCKD Stage 1- 2

Diabeties CKD Stage 3- 4

Diabeties CKD Stage 5(non dialyzed)

Diabeties Stage 5 (Hemodialysis)

Diabeties Stage 5 (Peritonealdialysis)

Adults ≥ 18 years ; non diabetics, with HBP, kaliemie>5.5 mmol/l, phosphoremie> 70 mg/l

HBPCKD Stage 1 (<60years)

HBP CKD Stage 1 (age ≥ 60 years)

HBP CKD Stage 2 (age ≥ 60 years)

HBP CKD Stage 2 (<60years)

HBP CKD Stage 3 (age ≥ 60

HBP CKD Stage 3 (<60years)

Adults ≥ 18 years ; non diabetics, with HBP, kaliemie< 5.5 mmol/l, phosphoremie<70 mg/l

HBPCKD Stage 1 (<60years)

HBP CKD Stage 1 (age ≥ 60 years)

HBP CKD Stage 2 (<60years)

HBP CKD Stage 2 (age ≥ 60 years)

HBP CKD Stage 3 (<60years)

HBP CKD Stage 3 (age ≥ 60

25 <eGFR≤ 29

HBP CKD Stage 4 (age ≥ 60

HBP CKD Stage 4 (<60years)

15<eGFR≤ 25

HBP CKD Stage 4 (age ≥ 60 years)

HBP CKD Stage 4 (<60years)

CKD stage 5 (Non dialyzed)

HBP CKD Stage 5 (<60years)

HBP CKD Stage 5 (age ≥ 60

CKD stage 5 (dialyzed)

HBPHemodialysis

HBP Peritonealdialysis

Adults ≥ 18 years ; diabetics, with HBP

Diab+HBPCKD Stage 1- 2

Diab+HBP CKD Stage 3- 4

Diab+HBP CKD Stage 5(non dialyzed)

Diab+HBPStage 5 (Hemodialysis)

Diab+HBP Stage 5 (Peritoneal dialysis)

Children (≤ 18 years)

0-6months (pre-dialysis high phosphorus)

0-6months (pre-dialysis normal phosphorus)

0-6months (hemodialysis, normal phosphorus)

0-6months (hemodialysis, high phosphorus)

0-6months (peritoneal dialysis, normal phosphorus)

0-6months (peritoneal dialysis, high phosphorus)

7-12 months (pre-dialysis, high phosphorus)

7-12 months (pre-dialysis, normal phosphorus)

7-12 months (hemodialysis, high phosphorus)

7-12 months (hemodialysis, normal phosphorus)

7-12 months (peritoneal dialysis, normal phosphorus)

7-12 months (peritoneal dialysis, high phosphorus)

1 to 3 years : Girls (see boys in the other document)

1-3 years (pre-dialysis; normal phosphorus)

1-3 years (pre-dialysis; high phosphorus)

1-3 years (hemodialysis; high phosphorus)

1-3 years (hemodialysis; normal phosphorus)

1-3 years (peritoneal dialysis; high phosphorus)

1-3 years (peritoneal dialysis; normal phosphorus)

4 to 6 years : Girls (see boys in the other document)

4-6 years (pre-dialysis, high phosphorus)

4-6 years (pre-dialysis, normal phosphorus)

4-6 years (hemodialysis, high phosphorus)

4-6 years (hemodialysis, normal phosphorus)

4-6 years (peritoneal dialysis, high phosphorus)

4-6 years (peritoneal dialysis, normal phosphorus)

7 to 10 years : Girls (see boys in the other document)

7-10 years (predialysis, high phosphorus)

7-10 years (predialysis, normal phosphorus)

7-10 years (hemodialysis, high phosphorus)

7-10 years (hemodialysis, normal phosphorus)

7-10 years (peritoneal dialysis, high phosphorus)

7-10 years (peritoneal dialysis, normal phosphorus)

GIRLS

11-14 years (predialysis, normal phosphorus)

11-14 years (predialysis, high phosphorus)

11-14 years (hemodialysis, normal phosphorus)

11-14 years (hemodialysis, high phosphorus)

11-14 years (peritoneal dialysis, high phosphorus)

11-14 years (peritoneal dialysis, normal phosphorus)

Boys

11-14 years (predialysis, high phosphorus)

11-14 years (predialysis, normal phosphorus)

11-14 years (hemodialysis, high phosphorus)

11-14 years (hemodialysis, normal phosphorus)

11-14 years (peritoneal dialysis, high phosphorus)

11-14 years (peritoneal dialysis, normal phosphorus)

Girls

15-18 years (predialysis, high phosphorus)

15-18 years (predialysis, normal phosphorus)

15-18 years (hemodialysis, high phosphorus)

15-18 years (hemodialysis, normal phosphorus)

15-18 years (peritoneal dialysis, high phosphorus)

15-18 years (peritoneal dialysis, normal phosphorus)

Boys

15-18 years (predialysis, high phosphorus)

15-18 years (predialysis, normal phosphorus)

15-18 years (hemodialysis, high phosphorus)

15-18 years (hemodialysis, normal phosphorus)

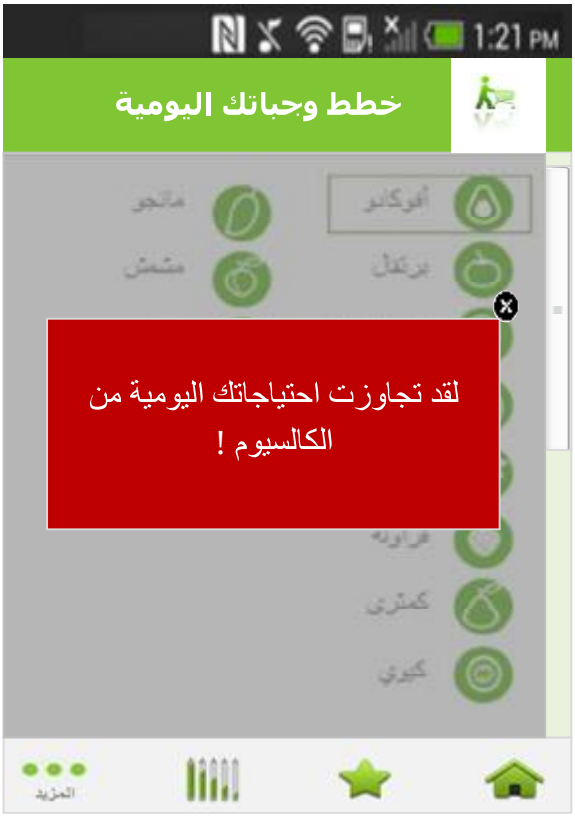
15-18 years (peritoneal dialysis, high phosphorus)

15-18 years (peritoneal dialysis, normal phosphorus)

Left for the Doctorrecommendations


1:21 PM
المبادئ التوجيهية الغذائية
التوصيات الغذائية المناسبة لك
البروتينات
الصوديوم
البوتاسيوم
الكلسيوم
الفوسفور
السوائل
السعرات الحرارية
المزيد

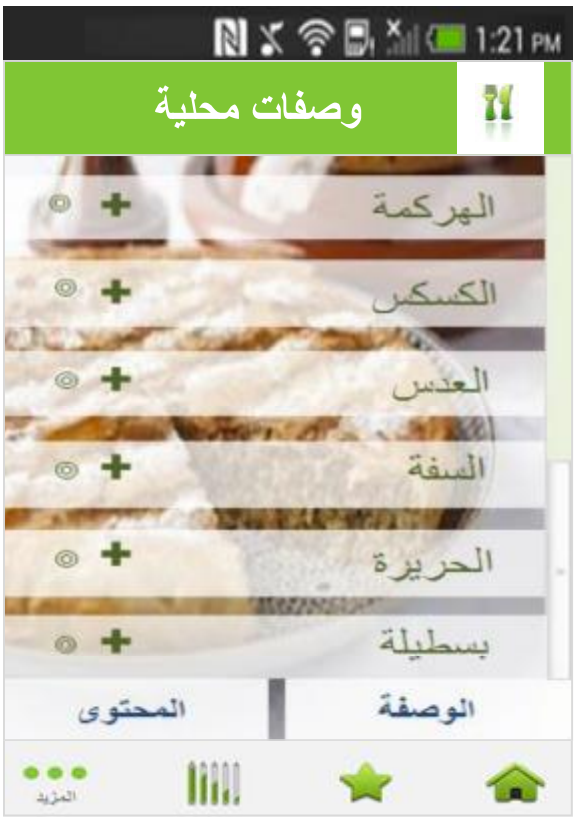
خطط وجباتك اليومية

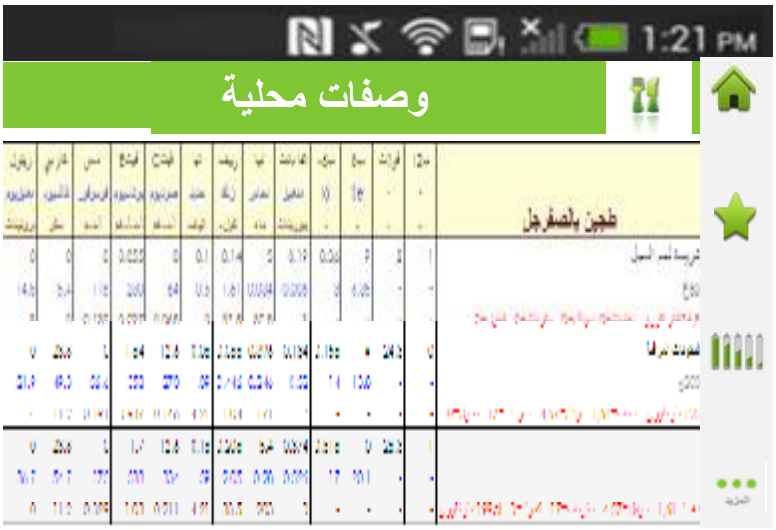

1:21 PM
خطط وجباتك اليومية
لقد تجاوزت احتياجاتك اليومية من الكالسيوم !
المزيد


1:21 PM
خطط وجباتك اليومية
إغلاق و إلغاء
الرجاء تحديد الكمية
0,5
100غ
250غ
500غ
أضف
المزيد

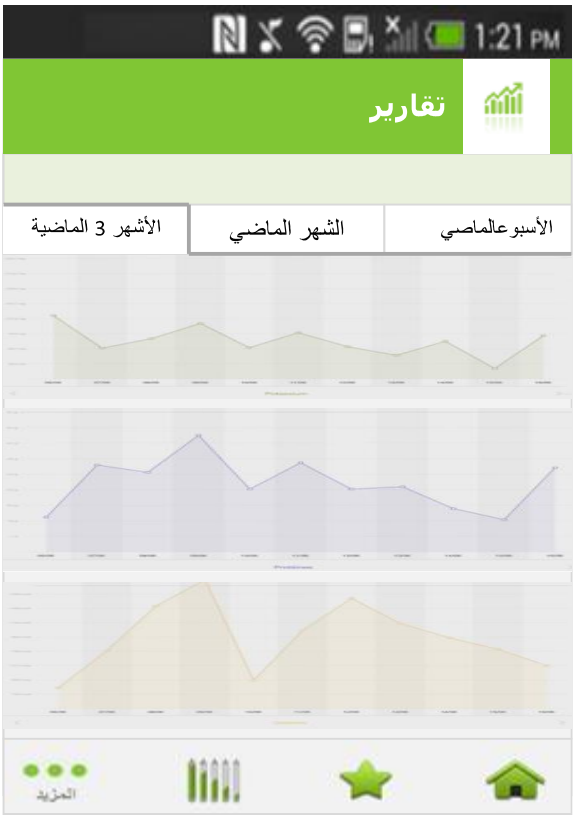
وصفات محلية


1:21 PM
وصفات محلية
السلطة المغربية
المكونات :
- 1 فلفل أخضر
- حبتي طماطم
- 1 خيار
- 1 بصلة صغيرة
- ملح، فلفل أسود
- 1 ملعقة زيت الزيتون أو زيت السلطة الثلاثة
- 1 ملعقة صغيرة من الخل
للتزيين
- بعض الزيتون الأسود
الطريقة
المزيد

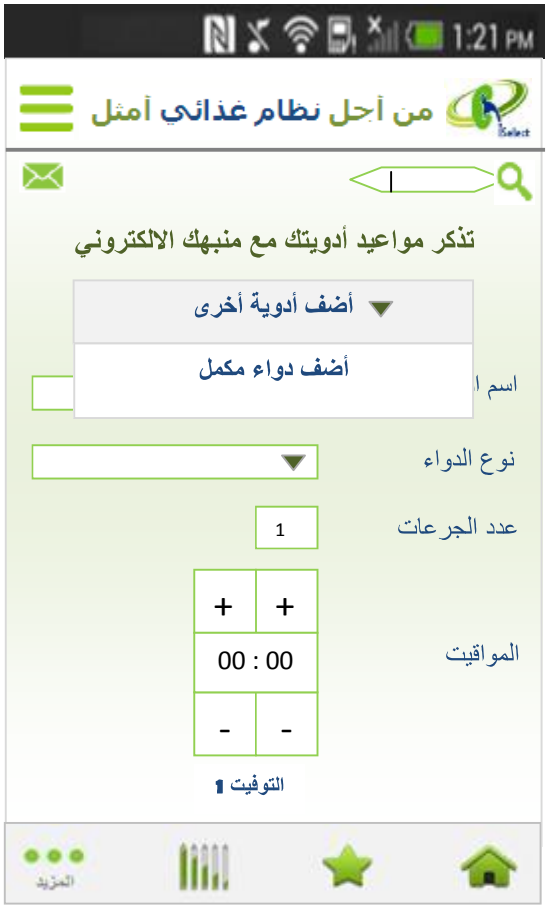

1:21 PM
وصفات محلية
الهركمة
الكسكس
العدس
السفة
الحريرة
بسطيلة
المحتوى
الوصفة
المزيد

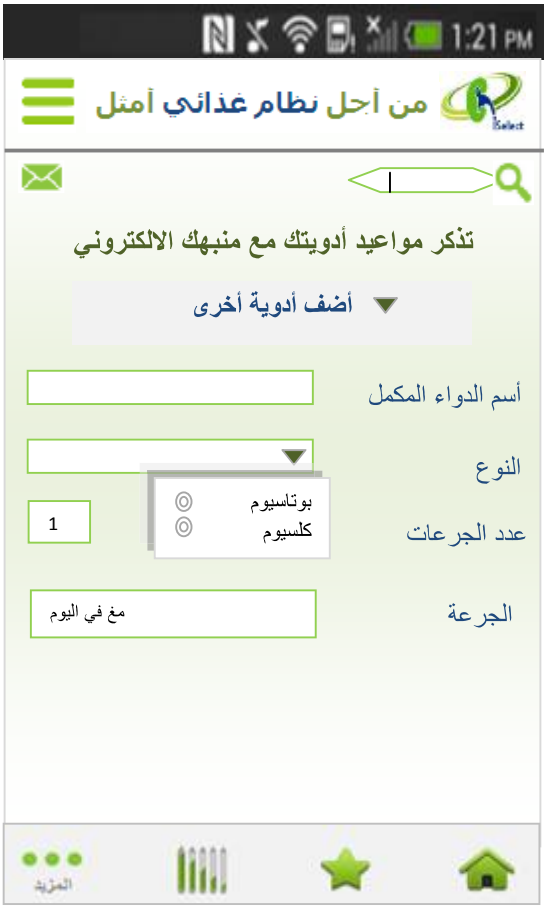

1:21 PM
وصفات محلية
المزيد

تقارير


1:21 PM
تقارير
الأسبوعالماضي
الشهر الماضي
الأشهر 3 الماضية
المزيد

المنبه الالكتروني





Survey for a better nutritional management in chronic kidney failure

---

Are you :

- **Male**
- **Female**

- **Adult (>15 years)**
- **Child**

- **Educated**
- **Illiterate**

Your CKD stage is :

- **Stage 1**
- **Stage 2**
- **Stage 3**
- **Stage 4**
- **Stage 5**

- **Hemodialysis**
- **Peritoneal dialysis**

Do you find it difficult to follow the diet prescribed by your Nephrologist ?

- **A lot**
- **A little bit**
- **Not really**

At the end of the day, do you come to reach your nutritional goals ?

- **Often**
- **Rarely, I get lost**

What is your nutritional behavior, on Moroccan special occasions (Ramadan, Adha feast, marriage ceremonies…)?

- **I keep following my diet**
- **I lose control**

Does those difficulties affect your life quality and well-being ?

- **Yes**
- **No**

What do you do when you can't manage your intakes according to your Doctor's recommendations ?

- **I let down the diet**
- **I keep trying**
- **I see my Doctor/Dietitian**

Do you take any pharmaceuticals ?

- **Yes (>2 drugs)**
- **Less**

Do you often forget your drugs schedule?

- **Yes**
- **Some times**
- **Never**

Do you see that the creation of a self-nutritional-manage tool would be very helpful ?

- **Absolutely**
- **I can manage on my own**

If this tool could help you further, to remember taking your medicines, would you be ready to apply it ?

- **Yes I'm in**
- **I would try to**
- **No**

Leave here please your own comments or propositions:

TABLE DE COMPOSITION DES ALIMENTS

Aliments (100 g)	kJ	eau	Prot.	Gluc.	G. S	G C.	Fib.	Lip.	AGSat	AG MI	AG PI	CS	Na	Mg	P	K	Ca	Fe	vit. A	vit. E	Vit. C	Vit. B1	Vit. B2	vit. PP	vit. B5	vit. B6	vit. B12	vit. B9
Aliments (100 g)	Valeur énergétique métabolisable	Eau	Protides	Glucides disponibles	Glucides simples	Glucides complexes	Fibres Alimentaires	Lipides	Acides Gras saturés	AG monoinsaturés	AG poly insaturés	Cholestérol	Na	Mg	P	K	Ca	Fe	rétinol et équivalent β carotène	Tocophérol	Ac. Ascorbique	Thiamine	Riboflavine	Niacine	Acide pantothénique	Pyridoxal	Cyano cobalamine	Acide folique
	kJ	%	en g									en mg							en ER	en mg							en µg	
																			Vit. A	Vit. E	Vit. C	Vit. B1	Vit. B2	Vit. PP	Vit. B5	Vit. B6	Vit. B12	Vit. B9
Viandes - Volailles																												
agneau, côtelette, crue	866	68	15	0	0	0	0	16,5	8	6,3	0,77	78	75	16	170	320	9	2	0	0,15	0	0,13	0,18	4,3	1	0,2	1,5	3
agneau, côtelette, grillée	976	61	22,6	0	0	0	0	16	7,8	6,1	0,75	83	90	17	177	333	9	2,4	0	0,2	0	0,1	0,21	5	1	0,2	1,7	3
bœuf, entrecôte, grillée	850	62	24,3	0	0	0	0	11,8	5	5,6	0,47	70	50	21	180	320	8	2,6	0	0,4	0	0,07	0,3	6	1	0,3	2	16
bœuf, faux filet, grillé	700	64	28,1	0	0	0	0	6	2,6	2,7	0,27	60	60	25	240	400	6	3	0	0,3	0	0,09	0,3	4,5	0	0,4	2	15
bœuf, flanchet, cru	814	66	19,6	0	0	0	0	13	5,7	5,9	0,52	65	70	19	200	320	9	2,5	0	0,3	0	0,08	0,2	4,1	1	0,3	2	9
bœuf, flanchet, cuit	966	57	29,4	0	0	0	0	12,6	5,3	6	0,5	80	52	19	170	250	17	3,5	0	0,4	0	0,06	0,3	3	1	0,3	2	7
bœuf, rosbif, rôti	628	66	28	0	0	0	0	4,1	1,7	1,9	0,16	55	65	25	230	400	5	3,5	0	0,5	0	0,08	0,24	5	1	0,4	2	14
porc, côtelette, crue	878	65	19	0	0	0	0	15	5,8	6,8	1,3	80	69	21	166	285	9	1,3	0	0,1	m	0,74	0,19	4,2	1	0,43	1,2	4
porc, côtelette, grillée	1031	56	28	0	0	0	0	15	5,8	6,8	1,3	84	72	24	220	400	11	1,1	0	0	0	0,59	0,24	5,7	1	0,3	0,8	6
porc, filet, maigre, cru	475	74	21	0	0	0	0	3,2	1,3	1,5	0,28	65	125	25	230	420	8	1,2	0	0,1	m	1	0,26	4,3	1	0,45	0,7	4
porc, filet, rôti, maigre	667	65	28,8	0	0	0	0	4,8	1,7	2,2	0,58	73	65	25	290	540	9	1,5	0	0,1	0	0,9	0,4	4,7	1	0,4	0,6	6
veau, filet, cru	458	75	20,4	0	m	m	0	3	0,95	1,1	0,37	80	92	25	210	328	16	0,8	0	0,15	0	0,08	0,26	8,6	1	0,54	1,2	14
veau, filet, rôti	675	65	28,4	0	0	0	0	5,2	1,8	2	0,62	98	93	25	236	375	17	1,3	0	0,2	0	0,07	0,29	8,6	1	0,4	1,2	10
steak haché 15%, cru	850	65	18	0	m	m	0	14,7	6,2	7	0,59	63	62	19	186	300	9	2,3	0	0,7	0	0,08	0,2	3,9	0	0,38	2	10
steak haché 20%, cru	1044	61	17	0	0	0	0	20,4	8,6	9,7	0,82	69	68	18	140	270	7	1,8	0	0,19	0	0,06	0,18	4	0	0,35	2	8
steak haché 20%, cuit	1282	53	21	0	0	0	0	25	10,5	11,9	1	86	82	22	171	331	9	2,2	0	0,2	0	0,05	0,21	3,9	0	0,3	1,9	8
canard, rôti, viande	795	64	25	0	0	0	0	6	2,3	1,6	0,76	85	90	19	202	262	11	2,1	24	0	0	0,33	0,43	5,4	2	0,3	1,3	30
dinde, rôtie, viande	607	66	29,4	0	0	0	0	2,9	1	0,74	0,96	76	63	27	217	305	17	1,3	0	0	0	0,07	0,19	7	1	0,4	1,2	9
poule, avec peau, bouillie	1267	51	25,8	0	0	0	0	22,4	6,5	9	4,5	76	76	19	200	182	13	1,5	160	0,4	0	0,08	0,21	6,6	1	0,3	0,2	5
poulet, rôti	678	66	26,4	0	0	0	0	6,2	1,8	2,9	1,2	90	80	24	200	300	12	1,3	7	0,2	0	0,07	0,17	7,7	1	0,4	0,3	8
Abats - Charcuteries																												
lardons fumés crus	1236	53,2	16,2	0,56	0,5	0	0	25,7	9,62	11,9	2,94	57	1376	16	177	256	8,4	1	5,5	0,3	0,85	0,24	10,2	10,2	0,6	0,3	0,83	2
foie, génisse, cru	568	70	21,1	3,5	0	3,5	0	4	1,5	0,64	0,84	300	96	17	358	325	7	7,2	10250	0,5	25	0,26	2,9	14	8	0,8	81	266
foie, génisse, cuit	642	64	23,6	3,8	0	3,8	0	4,7	1,8	0,68	1	290	102	18	388	346	7	7,7	11033	0,5	20	0,2	3	12	7	0,7	67	254
jambon cuit supérieur, découenné dégraissé	474	73	21	0,4	0,4	0	0	3	1,1	1,4	0,36	50	786	21	212	280	7	1	0	0,18	11	0,9	0,2	6	0	0,5	0,3	30
jambon de Bayonne, cru, découenné et dégraissé	803	56	26,3	0,3	0,3	0	0	9,5	3,4	4,5	1	66	2700	22	230	250	9	1,4	0	0,2	13	1,2	0,3	8,7	1	0,6	0,5	2
boudin noir, cuit	1695	43	14	3	m	m	0	38	13,4	17,3	4,6	130	860	13	71	180	50	22	0	0,2	0	0,04	0,1	1,2	1	0	0,4	5
pâté de campagne	1358	52	14,3	2,4	1,5	0,9	0	29	11	13	3,3	134	710	19	231	233	15	5,7	4200	0,3	6	0,31	0,78	8,7	m	0,3	6	160
quenelle de volaille	822	66	6,8	15	1	14	m	12	m	m	m	m	515	10	74	86	37	0,8	20	0,37	0	0,04	0,09	1,1	m	0,1	m	m
saucisse de Strasbourg	1257	56	12,6	1	0	1	0	27,7	10,2	12,7	3,3	64	1000	10	173	100	37	1	0	0,25	0	0,3	0,2	2,4	1	0,1	0,5	2
saucisson sec	1758	33	26,3	1,6	0,1	1,5	0	34,7	12,9	15,5	4,2	70	2100	16	242	160	11	1,3	0	0,3	0	0,57	0,28	5,1	1	0,4	1,9	3

Aliments (100 g)	kJ	eau	Prot.	Gluc.	G. S	G C.	Fib.	Lip.	AGSat	AG MI	AG PI	CS	Na	Mg	P	K	Ca	Fe	vit. A	vit. E	Vit. C	Vit. B1	Vit. B2	vit. PP	vit. B5	vit. B6	vit. B12	vit. B9
Poissons - Crustacés - Mollusques																												
cabillaud, (morue), au four	413	76	22,1	0	0	0	0	1	0,2	0,14	0,4	58	210	34	164	300	18	0,4	0	0,6	0	0,08	0,07	2,1	0	0,3	1,5	12
cabillaud, (morue), cru	333	80	18,1	m	m	m	0	0,7	0,12	0,1	0,3	43	76	25	180	340	16	0,1	7	0,5	m	0,06	0,06	2,4	0	0,22	0,96	12
crevette rose, cuite	437	73	21,8	0	0	0	0	1,8	0,3	0,37	0,6	185	1595	69	215	221	115	3,3	0	1,5	0	0,02	0,02	1,5	0	0,1	1,9	5
maquereau, filet au vin blanc appertisé	864	66	16	0	0	0	0	16	3,6	6	4,1	70	515	25	35	235	20	2,2	37	m	m	0,03	0,24	5,7	1	0,18	7,5	5
moule, cuite	497	73	20,2	3,1	m	m	0	2,8	0,48	0,52	0,76	50	386	68	235	206	101	7,9	84	2,4	0	0	0,11	1	m	0,1	10,2	27
poisson pané, frit	972	56	14,8	15,3	0	15,3	0,7	12,2	2,4	4,4	4,5	40	415	25	110	260	20	0,7	0	m	0	0,09	0,11	1,6	0	0,2	1,4	18
roussette (saumonette) crue	565	71	18	0	0	0	m	7	1,2	2,1	2,1	63	100	35	220	230	20	0,9	130	m	m	0,11	0,18	2,9	1	m	3	2
roussette, frite	1016	57	17,7	7	m	m	0,2	16,3	3,8	7	4,4	60	207	33	207	238	46	1,1	0	2,1	0	0,07	0,1	4,2	m	m	1,2	m
sardine à l'huile	898	60	23	0	0	0	0	13,7	2,8	4,7	4,9	72	480	37	468	380	400	2,5	36	0,4	0	0,02	0,25	6,5	1	0,2	12	12
saumon cru élevage	794	64,7	19,5	0	0	0	0	12,5	2,99	4,32	3,72	60,3	42,3	23,7	197	303	8,7	0,5	2,63	2,81	<1	0,28	0,09	7,25	1,69	0,63	5,03	16,3
surimi en bâtonnets	347	76	12,6	6,1	0	6,1	0	0,7	m	m	m	35	700	14	60	64	13	0,3	0	m	0	0,02	0,04	0,21	m	0,02	1	m
thon, à l'huile, appert.	780	62	27,6	0	0	0	0	8,4	1,5	2,6	3,7	33	347	33	259	267	10	1,2	0	2	0	0,02	0,1	14	0	0,5	5	5
thon, naturel, appert.	494	72	25,6	0	0	0	0	1,6	0,51	0,38	0,46	54	415	28	182	277	9	1,6	0	0,9	0	0,02	0,07	11,2	0	0,4	3	7
Œufs																												
blanc d'œuf	187	88	10,5	0,3	0,3	0	0	0,1	0	0	0	0	160	10	15	142	6	0,1	0	0	0	0	0,44	0,1	0	0	0,1	12
jaune d'œuf	1449	50	16,5	0,2	0,2	0	0	31,5	9,4	12,3	4,1	1100	50	15	520	97	137	5,5	591	3,6	0	0,22	0,5	0	4	0,4	4,7	140
œuf entier, cru	606	76	12,5	0,3	0,3	0	0	10,5	3,1	4,2	1,3	380	133	11	188	125	55	1,8	207	1,2	0	0,08	0,46	0,1	2	0,1	1,6	60
Produits laitiers - Fromages																												
Bleu	1416	45	20,2	0	0	0	0	29	18,8	8	0,8	90	1150	27	350	178	722	0,6	140	0,7	0	0,03	0,5	0,9	2	0,2	1,2	94
Camembert 45%	1176	54	21,2	0	0	0	0	22	13,8	6,4	0,6	60	802	18	309	110	400	0,2	393	0,5	0	0,05	0,6	1,1	1	0,3	2,8	96
Cantal	1520	42	23	0	0	0	0	30,5	19,3	8,9	0,7	90	940	30	570	136	970	0,4	221	0,5	0	0,04	0,3	0,1	0	0,1	1,5	21
Emmental	1572	38	29,4	0,1	0,1	0	0	28,8	17,3	8,9	1	110	226	45	746	98	1185	0,8	266	0,4	0	0,05	0,34	0,1	0	0,1	2,2	9
fromage chèvre, sec	1927	31	27,6	0	0	0	0	39,4	25,4	10,6	1,4	100	790	26	796	114	190	1,1	0	m	0	0,14	1,2	2,4	m	1,2	m	53
crème de gruyère	1429	52	11	6	6	0	0	19	12	6	0,9	100	650	10	215	100	102	m	0	0,6	0	m	m	m	m	0,1	0,3	m
fromageblanc 20%	337	84	8,3	3,6	3,6	0	0	3,4	2,2	1	0,1	10	33	11	60	120	117	0,4	52	0,1	1	0,04	0,27	0,1	1	0,1	0,8	16
fromage blanc 40%	479	81	7	3,4	3,4	0	0	8	5,1	2,3	0,3	30	29	10	93	90	109	0,3	93	0,3	1	0,03	0,24	0,1	1	0,1	0,7	26
Gouda	1437	42	24,9	0	0	0	0	27,4	17,7	7,8	0,66	110	620	29	490	114	854	0,4	359	0,53	0	0,03	0,28	0,07	0	0,07	1,7	21
lait écrémé en poudre	1494	4	35,5	49,5	49,5	0	0	0,8	0,52	0,21	0	3	682	112	1106	1537	1301	0,5	0	0	6	0,38	1,8	1	4	0,25	3	43
lait entier concentré	546	75	6,4	9,2	9	0	0	7,5	4,7	2,3	0,18	30	138	24	201	234	255	0,2	91	0,27	1	0,08	0,33	0,19	1	0,05	0,19	8
lait entier concentré sucré	1372	25	8,4	53,1	53,1	0	0	9,1	5,8	2,3	0,22	30	128	27	230	370	280	0,2	113	0,17	3	0,09	0,42	0,22	1	0,05	0,5	11
lait UHT, demi écrémé	195	90	3,2	4,6	4,6	0	0	1,6	1	0,5	0	7	46	10	85	166	114	0,1	23	0,1	1	0,05	0,17	0,1	0	0	0,2	3
lait UHT, écrémé	145	91	3,3	4,6	4,6	0	0	0,2	0	0	0	2	45	11	88	174	112	0,1	0	0	1	0,05	0,16	0,1	0	0	0,2	3
lait UHT, entier	263	88	3,2	4,6	4,6	0	0	3,5	2,2	1,1	0,1	14	45	10	86	148	119	0,1	48	0,1	1	0,05	0,17	0,2	0	0	0,2	3
Parmesan	1587	29	35,7	0	0	0	0	26,5	16,7	7,7	0,6	80	913	46	782	113	1275	0,7	419	0,9	0	0,02	0,33	0,2	0	0,1	1,5	20
Petit Suisse 40%	590	76	9,4	3,3	3,3	0	0	10,1	6,4	2,9	0,3	20	31	10	90	110	111	0,2	120	m	1	0,03	0,3	0,1	m	0,1	0,7	29
Pont l'Evêque	1247	48	21,1	0	0	0	0	24	15,2	6,9	0,5	70	670	22	414	136	470	0,4	249	0,6	0	0	0,3	0,1	0	0,1	1,5	12
yaourt aux fruits, lait entier	477	74	3,5	18	18	0	0	2,7	1,7	0,8	0,1	10	55	13	100	206	130	0,2	40	0,1	2	0,05	0,23	0,1	0	0,1	0	3
yaourt nature	211	88	4,3	4,8	4,8	0	0	1,1	0,7	0,3	0	4	58	13	111	203	173	0,1	13	0	0	0,04	0,18	0,1	0	0	0	2

Aliments (100 g)	kJ	eau	Prot.	Gluc.	G. S	G C.	Fib.	Lip.	AGSat	AG MI	AG PI	CS	Na	Mg	P	K	Ca	Fe	vit. A	vit. E	Vit. C	Vit. B1	Vit. B2	vit. PP	vit. B5	vit. B6	vit. B12	vit. B9
Corps gras																												
beurre	3091	16	0,7	0,5	0,5	0	0	83	52,6	23,5	2	250	12	2	24	13	15	0,2	792	2	0	0	0,02	0	0	0	0	0
crème fraiche pasteurisée	1302	59	2,3	1,6	1,6	0	0	33,4	20,9	9,7	0,9	110	35	5	58	100	63	0,2	430	0,8	0	0,01	0,1	0,1	0	0	0	0
huile d' arachide	3696	0	0	0	0	0	0	99,9	19,8	45,2	30,1	0	0	0	0	0	0	0	0	17,2	0	0	0	0	0	0	0	0
huile d' olive	3696	0	0	0	0	0	0	99,9	14,5	71	10	0	0	0	0	0	0	0	0	12	0	0	0	0	0	0	0	0
huile de colza	3696	0	0	0	0	0	0	99,9	6,2	64,3	25,5	0	0	0	0	0	0	0	0	15	0	0	0	0	0	0	0	0
huile de tournesol	3696	0	0	0	0	0	0	99,9	11,6	22,5	61,4	0	0	0	0	0	0	0	0	56	0	0	0	0	0	0	0	0
margarine allégée	1555	57	0,7	0,5	0,5	0	0	41,5	13,3	8,8	17,9	1	100	0	8	7	12	0	0	6	0	0	0	m	0	0	0	0
margarine au tournesol	3071	16	0,8	0,3	m	m	0	82,5	14,1	31,2	33,2	m	118	2	20	38	27	0	95	43	0	0,01	0,03	m	0,1	0,01	m	1
pâte à tartiner allégée	1638	49	7,7	1	1	0	0	40,3	17,8	10,3	10,2	74	190	8	280	80	23	m	0	m	m	m	m	m	m	m	0	m
Produits amylacés – Biscuits et viennoiseries																												
biscotte	1648	6	10	73,6	3	70,6	4	5	1,4	1,8	1,1	0	350	18	130	160	42	1,3	0	1,2	0	0,05	0,06	1,3	0	0,1	0	m
biscuit à la cuillère	1337	26	9	60	41,4	18,6	m	4,4	1,3	1,5	0,64	180	160	13	145	124	31	2,1	100	0,49	0	0,09	0,17	0,73	m	0,13	1	m
biscuit type petit beurre	1847	3	8,2	75	20,5	54,5	2,2	10,9	6	3,5	0,8	33	312	18	97	142	32	1,1	55	0,4	0,1	0,11	0,09	1,5	m	0,2	0	14
cake (aux fruits)	1561	22	5,1	57	37,8	19,2	1	13,9	8	3,8	0,64	113	215	16	102	227	32	1,5	135	0,43	0	0,1	0,1	0,86	0	0,12	1	8
céréale sucrée pour petit déjeuner	1652	2,5	5,5	86,8	39,1	47,6	1,7	1,7	0,31	0,59	0,69	0	645	22	93	83	11	6,3	0	m	53	1,3	1,5	17,6	0,4	1,8	2,5	300
céréales chocolatées,enrichi	1630	3	8,7	79,8	32,1	47,7	3,9	4,3	2,17	1,25	0,45	0	442	30	85	210	331	7,3	0	1	25	1,13	1,27	14,3	5	16	0,8	160
croissant	1726	15	7,5	55	7,5	47,5	2,2	17,2	9,9	5,5	0,8	50	492	16	124	136	42	1,2	33	0,1	0	0,3	0,2	2,3	1	0,3	0	70
farine blanche	1476	13	10	71,5	1,5	70	3,5	1,3	0,2	0,1	0,6	0	3	20	120	135	16	1,2	0	0,3	0	0,1	0,05	0,6	0	0,2	0	24
fécule de maïs	1554	11	0,3	88	0	88	0,6	0	0	0	0	0	6	2	22	5	1	0,5	0	0	0	0	0	0	0	0	0	1
lentille, cuite	379	70	8,2	12,6	0,3	12,3	7,8	0,5	0,07	0,08	0,24	0	3	32	100	276	19	3,3	1	0	0	0,13	0,07	0,6	1	0,2	0	60
lentille, sèche	1339	10	24	50,4	1	49,4	11,2	1,2	0,2	0,2	0,45	0	24	100	300	700	50	8	13	m	m	0,5	0,25	2,2	2	0,6	0	200
maïs doux, appert.	410	73	3	18,2	6,9	11,3	2,3	1,2	0,2	0,3	0,6	0	304	22	69	200	4	0,6	18	0,45	1	0,04	0,08	1,4	0,5	0,09	0	33
Muesli (moyen)	1781	4,6	8,5	63	25,9	35,1	6,7	15,3	8,5	4,74	2,03	5,8	226	57,5	190	310	60,6	6,22	0,1	3,04	20,9	0,97	1,03	10,9	4,42	1,43	0,88	164
pain	1155	29	8	56	1,9	54	3,5	1	0,2	0,1	0,4	0	650	26	90	120	23	1,4	0	0,2	0	0,09	0,05	1	0	0,1	0	23
pain au chocolat	1708	22	7,4	46,4	m	m	2	20,7	m	m	m	50	588	25	m	140	28	m	0	m	0	m	m	m	m	m	0	m
pain de campagne	1113	30	9,1	54,4	1,9	52,5	3,5	0,9	0,15	0,07	0,43	0	786	22	m	126	22	m	0	0,18	0	0,09	0,05	1	0	0,12	0	23
pain de mie	1167	33	8	50,3	2	48,3	3,1	4	1	0,8	1,4	0	600	21	91	129	91	1,2	0	0,8	0	0,18	0,03	1,3	0	0	0	27
patate douce, crue	428	72	1,2	23	10,7	12,3	2,9	0,3	0,06	m	0,13	0	19	13	44	300	22	0,7	667	4	25	0,1	0,06	0,6	1	0,13	0	52
pâtes alimentaires, crues	1509	10	12,5	70,9	2,6	68,3	5	1,4	0,2	0,2	0,6	0	5	55	167	236	24	1,8	0	0	0	0,15	0,04	2,5	0	0,1	0	28
pétale de maïs au sucre, enri	1577	3	5	86	37	49	2,4	0,8	0,1	0,2	0,3	0	525	10	30	60	453	7,5	0	0,07	0	1,2	1,3	15	5	1,7	0,84	168
pois cassé, cuit	468	68	8,3	17,8	0,8	17	4,4	0,4	m	0,08	0,16	0	2	33	110	316	12	1,5	4	0,27	m	0,15	0,06	0,95	1	0,09	0	65
pois chiche, cuit	572	60	8,9	18,7	1,1	17,6	8,6	2,5	0,3	0,6	1,2	0	6	53	132	335	56	2,8	4	1,2	0	0,13	0,06	0,6	0	0,1	0	100
pomme de terre épluchée, crue	308	79	2,1	15,2	0,9	14,3	1,6	0,2	m	m	0,1	0	7	21	46	525	7	0,7	0	0,06	10	0,1	0,03	1	0,38	0,26	0	20
riz blanc, cru	1512	13	6,6	78,3	0	78,3	1,4	0,6	0,2	0,2	0,2	0	5	35	102	98	10	0,6	0	0,1	0	0,07	0,04	1,6	1	0,2	0	20
riz blanc, cuit	509	70	2,3	26,3	0	26,3	0,5	0,2	0	0	0	0	1	8	37	34	4	0,2	0	0	0	0,02	0,01	0,4	0	0,1	0	3
tapioca, cru	1525	13	0,5	85,7	0	85,7	0,4	0,2	0	0	0	0	4	3	20	20	11	1	0	0	0	0,01	0,01	0	0	0	0	0
Légumes																												
aubergine cuite	79	91,8	0,83	3,4	3,2	0,2	2,5	0,2	0,03	0,015	0,113	0	5,14	15	15	123	20,1	0,25	3,67	0,03	1,3	0,08	0,02	0,6	0,08	0,09	0	14
betterave cuite	177	88	2	8,1	7,6	0,5	2	0,14	0,02	0,03	0,05	0	26,2	16,2	20,1	232	17	0,59	3,66	0,15	4,3	0,02	0,03	0,22	0,12	0,05	0	38

Aliments (100 g)	kJ	eau	Prot.	Gluc.	G. S	G C.	Fib.	Lip.	AGSat	AG MI	AG PI	CS	Na	Mg	P	K	Ca	Fe	vit. A	vit. E	Vit. C	Vit. B1	Vit. B2	vit. PP	vit. B5	vit. B6	vit. B12	vit. B9
carotte, crue	132	89	0,8	6,6	6,4	0,2	2,6	0,3	0,05	0,02	0,12	0	35	10	16	286	27	0,3	1667	0,5	7	0,1	0,05	0,6	0	0,2	0	30
carotte, cuite	106	91	0,8	5	4,7	0,3	2,7	0,3	0,05	0,02	0,12	0	37	9	31	169	29	0,5	1467	0,5	2	0,06	0,02	0,2	0	0,1	0	22
champignon de Paris, appert.	66	92	2,3	0,5	m	m	2,5	0,5	m	m	m	0	344	12	69	116	23	0,8	0	m	2	0,02	0,19	2	2	0,06	0	10
chou-fleur, cru	89	92	2,4	2,3	2	0,3	2,4	0,3	0,05	0,02	0,15	0	14	15	48	319	20	0,5	7	0,17	50	0,1	0,07	0,6	1	0,22	0	83
concombre cru	41	96,9	0,59	1,5	1,38	0,08	0,7	0,16	0,01	0,00	0	0	2	12	21	145	14	0,2	5,16	0,1	3,2	0,03	0,03	0,04	0,24	0,05	0	14
courgette, crue	70	94	1,8	2	1,9	0,1	1	0,2	0,04	0,02	0,09	0	3	18	31	230	19	0,4	53	0	20	0,05	0,04	0,6	0	0,1	0	50
épinard, cru	74	92	2,7	0,8	0,7	0,1	2,6	0,4	0,08	m	0,2	0	65	58	52	529	104	2,7	674	1,8	40	0,1	0,22	0,7	0	0,2	0	192
haricot vert, cru	102	90	2,1	3,6	2,2	1,4	3,1	0,2	0,06	m	0,1	0	4	28	38	243	56	1	57	0,24	16	0,08	0,1	0,7	1	0,14	0	70
laitue, crue	52	95	1,2	1,3	1,3	0	1,5	0,3	0,04	0,01	0,16	0	15	11	24	234	37	0,3	60	0,5	8	0,08	0,07	0,4	0	0,1	0	84
navet pelé, cru	74	93	0,9	3,2	3	0,2	2	0,2	m	m	0,1	0	57	8	31	238	39	0,3	3	m	20	0,05	0,05	0,5	0	0,09	0	16
oignon cru	130	88,2	1,35	5,7	5,65	0	1,8	0,21	0,04	0,02	0,08	0	2,25	9,06	36	171	33	0,41	3	0,2	7,4	0,04	0,03	0,18	0,13	0,14	0	24
petit pois, appertisé	311	76	4,4	12,4	m	m	5	0,6	0,1	0,06	0,3	0	255	19	64	137	23	1,5	67	0,2	9	0,12	0,08	1	0	0,06	0	40
poireau, cru	99	91	1,6	3,7	3,5	0,2	2,8	0,3	m	m	0,2	0	12	11	35	256	31	0,9	83	0,73	18	0,07	0,04	0,4	0	0,3	0	96
potiron, pulpe, cru	88	93	0,6	4,5	3,9	0,6	1	0,1	0,05	0	0	0	1	7	20	274	18	0,4	200	0,1	5	0	0,07	0,5	0	0,07	0	25
tomate, crue	82	94	0,8	3,5	3,5	0	1,2	0,3	0	0	0,14	0	5	11	24	226	9	0,4	100	1	18	0,06	0,05	0,6	0	0,1	0	20
Fruits																												
abricot, frais	177	87	0,8	10	10	0	2,1	0,1	0	0	0	0	2	11	20	315	16	0,4	250	0,7	7	0,04	0,05	0,6	0	0,1	0	7
ananas, pulpe, frais	200	87	0,4	11,3	11,3	0	1,4	0,2	0	0	0,08	0	2	15	11	146	15	0,3	5	0,1	18	0,08	0,03	0,3	0	0,1	0	14
avocat	572	76	1,8	0,8	0,8	0	3	14,2	2,9	8,9	1,8	0	7	33	44	522	16	1	31	1,9	11	0,07	0,16	2	1	0,3	0	54
banane	379	74	1,1	21	17,2	3,8	2	0,3	0,12	0	0,06	0	1	30	22	385	8	0,4	11	0,3	12	0,04	0,07	0,6	0	0,5	0	23
fraise	142	90	0,7	7	7	0	2,2	0,5	0	0,07	0,26	0	2	12	23	152	20	0,4	7	0,2	60	0,02	0,03	0,5	0	0,1	0	62
kiwi	201	83	1,1	9,9	9,8	0,1	2,5	0,6	0	0	0	0	4	17	37	287	27	0,4	8	m	80	0,01	0,04	0,4	m	0,1	0	37
mangue, pulpe, fraîche	240	83	0,6	13,4	13,1	0,3	2,3	0,2	0,05	0,07	0,03	0	2	9	22	150	20	1,2	522	1,8	44	0,03	0,05	0,4	0	0,08	0	51
orange	178	87	1	8,6	8,6	0	1,8	0,2	0	0	0	0	4	10	16	179	40	0,1	20	0,2	53	0,09	0,04	0,3	0	0,1	0	30
pêche	177	87	0,5	10	10	0	2	0,1	0	0	0	0	1	8	19	160	10	0,4	83	0,5	7	0,02	0,05	1	0	0	0	16
poire	213	85	0,4	12,2	12,2	0	2,3	0,3	0,04	0,05	0,11	0	2	7	13	125	10	0,2	10	0,5	5	0,03	0,03	0,2	0	0	0	10
pomelo dit « pamplemousse »	126	90	0,7	5,9	5,9	0	1,3	0,1	0	0	0	0	1	9	12	141	19	0,2	3	0,3	37	0,04	0,02	0,3	0	0	0	14
pomme	210	85	0,3	11,7	11,6	0,1	2,1	0,3	0,06	0,02	0,1	0	3	4	9	120	5	0,2	12	0,5	5	0,03	0,02	0,1	0	0,1	0	13
pomme compote, conserve	324	78	0,2	19,1	19,1	0	1,6	0,1	0	0	0	0	3	10	9	61	4	0,3	6	m	2	0,02	0,03	0,1	0	0,1	0	4
prune, Reine Claude	223	82	0,8	12	12	0	2,3	0,2	0	0	0	0	1	8	25	243	13	0,4	30	0,5	5	0,05	0,03	0,5	0	0,1	0	10
raisin, sec	1139	16	2,6	65,8	65,8	0	6,7	0,5	0,16	0,14	0,14	0	23	31	85	783	40	2,4	2	0	4	0,11	0,14	0,9	0	0,2	0	9
Produits sucrés																												
cacao, poudre, sans sucre	1387	3	19,3	11,6	0	11,6	12,1	23,1	13,6	7,7	0,7	0	60	520	660	1920	130	12,5	0	0,4	0	0,13	0,25	2,7	1	0,1	0	30
chocolat à croquer	2161	1	4,5	57,8	53,3	4,5	5,9	30	17,8	9,6	0,9	1	15	112	173	365	50	2,9	6	0,5	0	0,06	0,1	0,5	0	0,1	0	6
confiture tout type	1127	30	0,5	68	68	0	1	0,1	0	0	0	0	16	6	14	105	12	0,5	8	0	5	0	0	0	0	0	0	2
poudre cacaotée sucrée	1602	3	4,45	84,8	81,6	3,2	6	2,3	1,25	0,7	0,07	0	220	91	140	1215	34,5	3,6	0	0,1	21	0,44	0,38	1,75	1,44	0,6	0,1	31
sucre blanc	1680	0	0	100	100	0	0	0	0	0	0	0	0	0	0	2	1	0,1	0	0	0	0	0	0	0	0	0	0

Valeurs Issues du Répertoire Général des aliments *2ème édition (éditeurs INRA éditions), CNVA-CIQUAL (Lavoisier TEC/DOC), Table CIQUAL 2008 site AFSSA.*
La Valeur énergétique métabolisable est l'énergie standard (STD) calculée selon la méthode de Greenfield et Southgate, incluant acides organiques, polyols…
La colonne Gluc.désigne les Glucides disponibles dont la valeur énergétique moyenne est de 17 kJ/g
La quantité de vitamine A résulte du calcul selon la formule = rétinol (µg) + 1/6 équivalent ß carotène (µg) sauf pour les produits laitiers (1/2 au lieu de 1/6)
m : *sont des valeurs manquantes ou bien à l'état de traces.*

COMPOSITION DE CERTAINS PRODUITS EN COMPLÉMENT A LA TABLE DE COMPOSITION DES ALIMENTS

PRODUITS	PROTIDES	LIPIDES	GLUCIDES	Na	K	Ca	OBSERVATIONS
	en g/100g ou g/100 mL suivant état			en mg pour 100 g			COMPLÉMENTAIRES
Lait sec écrémé non sucré (Régilait)	35,5	0,8	51,7	1 200	2 000	1 500	Sans saccharose
Lait croissance nature (Candia)	2,7	2,7	6,8	39	138	115	Fer = 1,4 mg
Lait Matin léger 1/2 écrémé (Lactel)	3,2	1,5	4,8	40		120	Lactose<0,5g
Lait sec DIARGAL (Gallia)	10.8	20,3	58.6	300	600	450	Sans lactose reconstitué à 13.3%
Lait sec AL 110 (Nestlé)	14	25	55,5	170	600	450	Sans lactose enrichi en fer 4,4 mg/100 g
Lait sec O-LAC (Mead-Johnson)	11	28	56	160	570	430	Sans lactose reconstitué à 13%
Nesvital poudre (Nestlé)	87,2	1,8	<0,3	60	NC	1 400	Protéines de lait
Protifar plus (Nutricia)	89	2	<1,5	NC	NC	NC	Goût neutre
Liprocil (Nestlé)	-	100	-	-	-	-	80 % T.C.M. + A.G.E.
Caloreen (Nestlé)	0	0	95	40	NC	NC	Dextrine-Maltose
Céréal Instant (Fresenius)	8,8	1,2	81,3	0	150	500	Préparation instantanée pour petit déjeuner
Clinutren poudre (Nestlé)	0	0	100	<200	NC	NC	Epaississant instantané à froid et à chaud
Fleur de Maïs (Maïzena)	0,4	0,6	87	4	4	NC	
Préparation de type farine pour pain et pâtisserie (Rite Diet)	0,3	0,3	90	8	26	NC	Hypoprotidique sans gluten
Préparation de type Biscotte (Aproten)	< 1	6	86	< 40	60	0	Hypoprotidique sans gluten
Pâtes à potage - spaghetti (Rite Diet)	0,5	1,5	85,5	< 100	< 20	40	Hypoprotidique sans gluten
Préparation de type pain de mie tranché (Valpiform)	4,5	3,9	48,1	NC	NC	NC	Sans gluten salé
Coquillettes (Schär)	9	2,5	73,6	100	_	_	Sans gluten
Légumes homogénéisés							
Haricots verts (Guigoz)	1,8	0,8	9,3	56	123	50	Sans sel ajouté
Jardinière de légumes (Guigoz)	1	0,5	9,8	130	113	19	
Carottes (Guigoz)	0,6	0,2	6	35	214	27	
Fruits homogénéisés							
Compote homogénéisée pomme-coing(Blédina)	0,3	0,2	12,2	10	NC	NC	10
Compote homogénéisée pommes-Bananes (Blédina)	0,3	0,3	14,3	10	NC	NC	10
Compléments oraux							
Fortimel Extra (Nutricia) Contenance:200mL	10	5,3	15,6	50	200	270	Parfum:vanille, chocolat, moka, fraise, abricots, fruits de la forêt, neutre
Fortimel sans lactose (Nutricia) Contenance:200mL	10	3,5	14,7	50	200	270	Parfum: vanille, chocolat, moka,caramel
Clinutren 1.5 (Nestlé) Contenance:200mL	5,5	10	21	80	170	80	Parfum: vanille, chocolat, fraise, café, banane, abricot
Ressource 2.0 fibres (Fresenius) Contenance:200mL	4,5	4,3	10,7	45	90	80	Parfum:vanille, chocolat, café, fruits des bois
Clinutren Fruit (Nestlé) Contenance:200mL	8	0	54	30	90 à 120	110	Parfum:Orange,pamplemousse, poire-cerise,framboise-cassis
Fortimel crème (Nutricia) Contenance: 125 ou 200g	10	5	19	NC	NC	NC	Vanille, chocolat, moka, banane, fruits de la forêt
Forti Yog (Nutricia) Contenance:200mL	6	5,8	18,7	NC	NC	NC	Texture yaourt vanille-citron, framboise, pêche-orange
Delical Potage HP (Lactalys NS) Contenance:200mL	7	4	9	NC	NC	NC	Carottes-tomates, poireaux-pommes de terre, légumes variés
Delical Mixé HP/HC (Lactalys NS) Contenance:300g	9	8,8	13,5	NC	NC	NC	Porc-jardinière, poulet-celeri, thon-courgettes

*** NC = non connu**

Composition des recettes en micronutriments

Hargma	Retol	CarBe	D-s	E	C	Thia
	Mg	Ca	P	K	Na	Fe
	ProAn	Sucr	AGsat	AGmi	AGpi	Fib-
Accompagnement De Harguma À La Marocaine	0.39	1392	0	4.07	42.4	0.178
200g	46.8	99.3	192	543	505	3.67
304kcal P:9.38 L:13.3 G:33.3 A:0	?	4.8	0.565	2.78	0.518	6.19
Veau, Escalope, Cuite	0	0	0	0.11	0	0.2
100g	29.5	10.8	236	460	128	1
149kcal P:31 L:2.75 G:0 A:0	?	0	0.277	0.194	0.13	0
	0.39	1392	0	4.18	42.4	0.378
	76.3	110	428	1003	633	4.67
453kcal P:40.4 L:16.1 G:33.3 A:0	0	4.8	0.842	2.97	0.647	6.19

Bakkoula	Retol	CarBe	D-s	E	C	Thia
	Mg	Ca	P	K	Na	Fe
	ProAn	Sucr	AGsat	AGmi	AGpi	Fib-
Bakkoula	0.81	10078	0	4.43	106	0.208
250g	109	325	110	1084	369	6.25
90.3kcal P:7.12 L:1.01 G:7.86 A:0	?	2.72	0.049	0.074	0.115	7.95
	0.81	10078	0	4.43	106	0.208
	109	325	110	1084	369	6.25
90.3kcal P:7.12 L:1.01 G:7.86 A:0	0	2.72	0.049	0.074	0.115	7.95

Bissara Fève	Retol	CarBe	D-s	E	C	Thia
	Mg	Ca	P	K	Na	Fe
	ProAn	Sucr	AGsat	AGmi	AGpi	Fib-
Bissara Fève	0	346	0	1.1	38.9	0.084
200g	42.2	44	215	473	729	2.2
139kcal P:10.8 L:1.75 G:15.4 A:0	?	3.57	0.072	0.056	0.152	9.29
	0	346	0	1.1	38.9	0.084
	42.2	44	215	473	729	2.2
139kcal P:10.8 L:1.75 G:15.4 A:0	0	3.57	0.072	0.056	0.152	9.29

Bissara pois cassé	Retol	CarBe	D-s	E	C	Thia
	Mg	Ca	P	K	Na	Fe
	ProAn	Sucr	AGsat	AGmi	AGpi	Fib-
Bissara Pois Cassé	0	86.6	0	0.544	3.5	0.282
200g	47.5	63.8	277	686	934	4.9
247kcal P:16.9 L:2.41 G:29.6 A:0	?	1.78	0.089	0.235	0.347	19.8
	0	86.6	0	0.544	3.5	0.282

	47.5	63.8	277	686	934	4.9
247kcal P:16.9 L:2.41 G:29.6 A:0	0	1.78	0.089	0.235	0.347	19.8

Coquelet au couscous	Retol Mg ProAn	CarBe Ca Sucr	D-s P AGsat	E K AGmi	C Na AGpi	Thia Fe Fib-
Coquelet Au Couscous	72.2	272	0.268	3.14	10.4	0.233
250g	69.1	90.8	323	559	492	3.81
623kcal P:34.5 L:40.3 G:28.2 A:0	?	10.1	2.8	5.32	2.32	3.85
	72.2	272	0.268	3.14	10.4	0.233
	69.1	90.8	323	559	492	3.81
623kcal P:34.5 L:40.3 G:28.2 A:0	0	10.1	2.8	5.32	2.32	3.85

Couscous au veau	Retol Mg ProAn	CarBe Ca Sucr	D-s P AGsat	E K AGmi	C Na AGpi	Thia Fe Fib-
Couscous Semoule Cuite À La Marocaine	28	17.7	0.035	2.94	0	0.338
250g	73.6	35.2	264	313	1196	1.82
520kcal P:8.86 L:15.8 G:83.2 A:0	?	0.253	1.05	2.71	0.304	3.26
Légumes De Couscous À La Marocaine	0.061	12	0	0.017	0.318	0.001
100ml (2.2g)	0.169	0.604	0.298	2.9	1.85	0.009
0.7kcal P:0.012 L:0.054 G:0.041 A:0	?	0.022	0.003	0.011	0.001	0.021
Veau, Escalope, Cuite	0	0	0	0.11	0	0.2
100g	29.5	10.8	236	460	128	1
149kcal P:31 L:2.75 G:0 A:0	?	0	0.277	0.194	0.13	0
	28	29.7	0.035	3.07	0.318	0.538
	103	46.6	500	776	1326	2.83
670kcal P:39.9 L:18.6 G:83.3 A:0	0	0.274	1.33	2.91	0.435	3.28

Lentille à la marocaine	Retol Mg ProAn	CarBe Ca Sucr	D-s P AGsat	E K AGmi	C Na AGpi	Thia Fe Fib-
Lentille À La Marocaine	0.1	822	0	3.64	22.4	0.164
200g	48.5	67.4	137	597	603	2.69
195kcal P:7.47 L:8.8 G:18.4 A:0	?	5.92	0.352	1.79	0.253	5.58
	0.1	822	0	3.64	22.4	0.164
	48.5	67.4	137	597	603	2.69
195kcal P:7.47 L:8.8 G:18.4 A:0	0	5.92	0.352	1.79	0.253	5.58

Rfissa au poulet	Retol Mg ProAn	CarBe Ca Sucr	D-s P AGsat	E K AGmi	C Na AGpi	Thia Fe Fib-
Mssamman À La Marocaine	0	0	0	6.99	0.292	0.374

200g	79.7	49.8	313	423	468	2.67
806kcal P:20.6 L:25.2 G:117 A:0	?	3.16	1.07	5.31	0.808	11
Sauce De Rfissa	0.402	4.92	0.002	0.032	0.152	0.002
150ml (1.7g)	0.594	0.73	2.58	4.08	2.31	0.024
4.5kcal P:0.303 L:0.345 G:0.035 A:0	?	0.023	0.02	0.048	0.023	0.028
Poulet, Cuisse, Viande, Bouilli	5	?	0.25	0.15	1.1	0.03
50g	13.1	2.9	82	131	45	0.6
95.5kcal P:0 L:0 G:0 A:0	?	0	0.337	0.451	0.223	0
	5.4	4.92	0.252	7.18	1.54	0.405
	93.4	53.4	397	558	515	3.29
906kcal P:20.9 L:25.5 G:117 A:0	0	3.19	1.43	5.81	1.05	11

Pastilla au poulet	Retol Mg ProAn	CarBe Ca Sucr	D-s P AGsat	E K AGmi	C Na AGpi	Thia Fe Fib-
Passtilla À La Marocaine	124	211	0.506	5.16	6.59	0.168
200g	77.1	118	290	426	472	2.49
631kcal P:28.5 L:42.4 G:30.3 A:0	?	14.7	3.03	5.89	2.45	4.21
	124	211	0.506	5.16	6.59	0.168
	77.1	118	290	426	472	2.49
631kcal P:28.5 L:42.4 G:30.3 A:0	0	14.7	3.03	5.89	2.45	4.21

Poulet beldi	Retol Mg ProAn	CarBe Ca Sucr	D-s P AGsat	E K AGmi	C Na AGpi	Thia Fe Fib-
Poulet Beldi	235	236	0.236	1.94	10.3	0.168
200g	33.8	40	257	360	268	2.8
502kcal P:33.2 L:34.2 G:14.6 A:0	?	2.41	2.22	4.53	2.06	1.38
	235	236	0.236	1.94	10.3	0.168
	33.8	40	257	360	268	2.8
502kcal P:33.2 L:34.2 G:14.6 A:0	0	2.41	2.22	4.53	2.06	1.38

Saffa aux cheveux d'anges	Retol Mg ProAn	CarBe Ca Sucr	D-s P AGsat	E K AGmi	C Na AGpi	Thia Fe Fib-
Saffa De Pates Alimentaires	17.5	12.2	0.022	1.26	0.298	0.19
200g	61.6	42	128	365	155	2.6
346kcal P:8.94 L:7.83 G:56.5 A:0	?	7.79	0.61	1.15	0.287	4.78
Accompagnement Saffa De Pates Alimentaires	0	1.27	0	6.21	0.464	0.079
50g	99.7	128	178	293	1.13	1.66
296kcal P:10.8 L:22.7 G:6.9 A:0	?	6.52	0.513	4.09	1.87	6.26
	17.5	13.5	0.022	7.47	0.762	0.269
	161	170	305	659	156	4.26

642kcal P:19.8 L:30.5 G:63.4 A:0	0	14.3	1.12	5.24	2.16	11

Saffa au riz	Retol Mg ProAn	CarBe Ca Sucr	D-s P AGsat	E K AGmi	C Na AGpi	Thia Fe Fib-
Saffa De Riz	57.2	36.1	0.072	0.108	0	0.096
200g	22	16.8	74.2	58.6	224	0.734
319kcal P:4.8 L:7.65 G:56.9 A:0	?	2.26	1.31	0.431	0.099	2.1
Accompagnement Saffa De Riz	0	1.27	0	6.21	0.464	0.079
50g	99.7	128	178	293	1.13	1.66
296kcal P:10.8 L:22.7 G:6.9 A:0	?	6.52	0.513	4.09	1.87	6.26
	57.2	37.4	0.072	6.32	0.464	0.175
	122	145	252	352	225	2.4
615kcal P:15.6 L:30.3 G:63.8 A:0	0	8.78	1.83	4.52	1.97	8.36

Tajine viande au cardon	Retol Mg ProAn	CarBe Ca Sucr	D-s P AGsat	E K AGmi	C Na AGpi	Thia Fe Fib-
Accompagnement Tajine De Viande Au Cardon	0	6.1	0	1.42	6.85	0.052
200g	37.9	97.3	52.1	644	280	0.79
85.1kcal P:1.67 L:4.23 G:8.44 A:0	?	3.76	0.179	0.891	0.113	3.26
Veau, Escalope, Cuite	0	0	0	0.055	0	0.1
50g	14.8	5.4	118	230	64	0.5
74.5kcal P:0 L:0 G:0 A:0	?	0	0.139	0.097	0.065	0
	0	6.1	0	1.48	6.85	0.152
	52.7	103	170	874	344	1.29
160kcal P:1.67 L:4.23 G:8.44 A:0	0	3.76	0.317	0.988	0.178	3.26

Tajine viande au coing	Retol Mg ProAn	CarBe Ca Sucr	D-s P AGsat	E K AGmi	C Na AGpi	Thia Fe Fib-
Veau, Escalope, Cuite	0	0	0	0.055	0	0.1
50g	14.8	5.4	118	230	64	0.5
74.5kcal P:0 L:0 G:0 A:0	?	0	0.139	0.097	0.065	0
Accompagnement Tajine De Viande Au Coing	0	23.8	0	1.64	12.6	0.06
200g	21.9	49.3	56.6	358	270	1.09
125kcal P:1.61 L:4.57 G:17 A:0	?	11.2	0.191	0.937	0.146	4.21
	0	23.8	0	1.7	12.6	0.16
	36.7	54.7	175	588	334	1.59
199kcal P:1.61 L:4.57 G:17 A:0	0	11.2	0.329	1.03	0.211	4.21

	Retol Mg	CarBe Ca	D-s P	E K	C Na	Thia Fe

Tajine viande au courgette	ProAn	Sucr	AGsat	AGmi	AGpi	Fib-
Veau, Escalope, Cuite	0	0	0	0.055	0	0.1
50g	14.8	5.4	118	230	64	0.5
74.5kcal P:0 L:0 G:0 A:0	?	0	0.139	0.097	0.065	0
Accompagnement Tajine De Viande Au Courgette	0.036	526	0	1.96	25.5	0.122
200g	33.3	42.4	54.4	365	185	1.17
60.9kcal P:1.99 L:3.13 G:5.1 A:0	?	3.26	0.135	0.615	0.094	2.13
	0.036	526	0	2.02	25.5	0.222
	48.1	47.8	172	595	249	1.67
135kcal P:1.99 L:3.13 G:5.1 A:0	0	3.26	0.274	0.712	0.158	2.13

Tajine viande au haricot vert et au courgette	Retol Mg ProAn	CarBe Ca Sucr	D-s P AGsat	E K AGmi	C Na AGpi	Thia Fe Fib-
Veau, Escalope, Cuite	0	0	0	0.055	0	0.1
50g	14.8	5.4	118	230	64	0.5
74.5kcal P:0 L:0 G:0 A:0	?	0	0.139	0.097	0.065	0
Accompagnement Tajine De Viande Au Haricat Vert Et Au Courge	0.05	678	0	1.99	24.4	0.116
200g	37.7	71.2	69.3	416	348	1.33
91.2kcal P:2.52 L:4.31 G:8.39 A:0	?	4.46	0.19	0.874	0.132	3.98
	0.05	678	0	2.04	24.4	0.216
	52.4	76.6	187	646	412	1.83
166kcal P:2.52 L:4.31 G:8.39 A:0	0	4.46	0.329	0.971	0.197	3.98

Tajine viande au petit pois et artichaut	Retol Mg ProAn	CarBe Ca Sucr	D-s P AGsat	E K AGmi	C Na AGpi	Thia Fe Fib-
Veau, Escalope, Cuite	0	0	0	0.055	0	0.1
50g	14.8	5.4	118	230	64	0.5
74.5kcal P:0 L:0 G:0 A:0	?	0	0.139	0.097	0.065	0
Accompagnement Tajine De Viande Au Petits Pois Et Artichaut	0.038	421	0	1.35	15.7	0.068
200g	58.9	72.2	87.9	598	296	1.44
108kcal P:4.97 L:3.51 G:10 A:0	?	3.72	0.159	0.672	0.128	7.6
	0.038	421	0	1.41	15.7	0.168
	73.6	77.6	206	828	360	1.94
182kcal P:4.97 L:3.51 G:10 A:0	0	3.72	0.298	0.77	0.193	7.6

Tajine viande au pruneau	Retol Mg ProAn	CarBe Ca Sucr	D-s P AGsat	E K AGmi	C Na AGpi	Thia Fe Fib-
Veau, Escalope, Cuite	0	0	0	0.055	0	0.1
50g	14.8	5.4	118	230	64	0.5

74.5kcal P:0 L:0 G:0 A:0	?	0	0.139	0.097	0.065	0
Accompagnement Tajine De Viande Au Pruneau À La Marocaine	13.7	210	0.018	4.99	3.93	0.12
200g	71.2	110	149	614	346	2.27
327kcal P:6.77 L:16.3 G:33.2 A:0	?	24.1	0.728	2.92	0.921	6.84
	13.7	210	0.018	5.05	3.93	0.22
	86	115	267	844	410	2.77
402kcal P:6.77 L:16.3 G:33.2 A:0	0	24.1	0.867	3.02	0.986	6.84

Harrira	Retol Mg ProAn	CarBe Ca Sucr	D-s P AGsat	E K AGmi	C Na AGpi	Thia Fe Fib-
Harira Marocaine	17	1247	0.023	1.81	33.6	0.278
250g	60	88	255	788	468	3.13
317kcal P:21.8 L:3.93 G:44.7 A:0	?	5.12	0.514	0.295	0.303	6.63
	17	1247	0.023	1.81	33.6	0.278
	60	88	255	788	468	3.13
317kcal P:21.8 L:3.93 G:44.7 A:0	0	5.12	0.514	0.295	0.303	6.63

Salade marocaine	Retol Mg ProAn	CarBe Ca Sucr	D-s P AGsat	E K AGmi	C Na AGpi	Thia Fe Fib-
Salade Marocaine	0	607	0	3.65	55.4	0.092
200g	25.4	32.1	39.9	364	239	0.648
135kcal P:1.6 L:10.9 G:5.53 A:0.002	?	4.22	0.437	2.28	0.272	3.33
	0	607	0	3.65	55.4	0.092
	25.4	32.1	39.9	364	239	0.648
135kcal P:1.6 L:10.9 G:5.53 A:0.002	0	4.22	0.437	2.28	0.272	3.33

Maâkouda	Retol Mg ProAn	CarBe Ca Sucr	D-s P AGsat	E K AGmi	C Na AGpi	Thia Fe Fib-
Maâkouda À La Marocaine	40.7	499	0.316	4.78	25.3	0.2
200g	39.9	66	121	545	161	2.58
335kcal P:7.31 L:19.9 G:30.3 A:0	?	1.68	0.89	4.11	0.516	3.96
	40.7	499	0.316	4.78	25.3	0.2
	39.9	66	121	545	161	2.58
335kcal P:7.31 L:19.9 G:30.3 A:0	0	1.68	0.89	4.11	0.516	3.96