



Beyond Body Mass Index in Colorectal Cancer: Body Composition, Metabolic Phenotypes, and Precision Risk Stratification

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Abstract

Background: Body mass index (BMI) is the most widely used measure of adiposity in oncology, yet it cannot distinguish visceral fat from subcutaneous fat, skeletal muscle from adipose tissue, or stable obesity from cancer-associated weight loss. These limitations are clinically important in colorectal cancer (CRC), where apparently favorable outcomes among some overweight patients have contributed to the so-called obesity paradox. **Aim:** To examine how body composition and metabolic phenotype can refine prognostic assessment beyond BMI in CRC and to define practical implications for surgery, systemic therapy, survivorship, and precision risk stratification. **Methods:** Clinical cohorts, meta-analyses, body-composition studies, treatment-dosing guidance, and survivorship evidence published through 2024 were synthesized. **Results:** BMI shows nonlinear associations with CRC outcomes and is vulnerable to confounding by smoking, comorbidity, reverse causation, and prediagnostic weight loss. Computed tomography-derived visceral adiposity, skeletal-muscle index, muscle attenuation, and sarcopenic obesity provide biologically richer information. Low muscle mass and myosteatosis are consistently associated with postoperative morbidity, treatment toxicity, and poorer survival, while visceral obesity can increase technical surgical risk and may coexist with apparently normal BMI. Full weight-based chemotherapy dosing remains recommended in obesity. Longitudinal weight change, nutrition, inflammation, diabetes, and physical activity further modify risk. **Conclusion:** Precision assessment of the CRC host should move from a single BMI value toward integrated body-composition and metabolic phenotyping. Routine staging CT creates an opportunity to obtain these measures without additional imaging, potentially improving prognostic modeling and individualized supportive care.

Keywords: Colorectal cancer, Body composition, Sarcopenia, Visceral adiposity, Obesity paradox

Introduction

Body mass index remains the dominant measure used to classify overweight and obesity in clinical oncology because it is inexpensive, reproducible, and universally available. Its apparent simplicity is also its major weakness. BMI measures total body mass relative to height but does not identify how much of that mass is visceral fat, subcutaneous fat, skeletal muscle, edema, or tumor-associated fluid. In CRC, where weight loss and muscle depletion may begin before diagnosis, the same BMI can represent very different biological states. [1]

The epidemiologic link between excess body weight and CRC incidence is well established. A large meta-analysis showed increased risk in obesity, and subsequent work has confirmed that abdominal adiposity is particularly relevant. Prognostic associations after diagnosis are less consistent. Some studies report worse survival with obesity, others find little difference, and several suggest the lowest mortality among overweight rather than normal-weight patients. This pattern has been labeled the obesity paradox but may partly reflect limitations in exposure measurement and study design. [2]

Modern staging CT contains far more host information than conventional BMI. A single axial image can quantify skeletal muscle, visceral adipose tissue, subcutaneous adipose tissue, and muscle attenuation. These measures identify sarcopenia, visceral obesity, and myosteatosis, all of which have been associated with clinically meaningful outcomes in CRC. Because the images already exist for oncologic staging, body-composition analysis has the potential to become an opportunistic biomarker rather than an additional test. [3]

Research Gap

Clinical decision-making still relies heavily on BMI despite increasing evidence that muscle depletion, fat distribution, metabolic health, and weight trajectory carry distinct prognostic information. The unresolved issue is not whether BMI should be discarded, but how it should be integrated with imaging-derived body composition, metabolic comorbidity, inflammation, treatment



exposure, and longitudinal change to produce clinically useful risk models.

Aim

To review the limitations of BMI in colorectal cancer and evaluate how visceral adiposity, skeletal muscle, myosteatosis, sarcopenic obesity, weight trajectory, and metabolic health may improve prognostic and therapeutic risk stratification.

Why BMI Is an Imperfect Oncologic Biomarker

BMI cannot distinguish fat from lean tissue. A muscular patient and a patient with high adiposity may share the same BMI, while an older or cachectic patient can have a normal BMI despite severe muscle depletion. This misclassification is especially relevant in oncology because skeletal muscle is a functional organ that influences mobility, insulin sensitivity, amino-acid reserve, drug distribution, and recovery from surgical or systemic stress. A BMI-only approach therefore compresses biologically different phenotypes into the same category. [4]

BMI also ignores fat distribution. Visceral adipose tissue is more strongly associated than subcutaneous fat with insulin resistance, inflammatory cytokines, dyslipidemia, and hepatic metabolic dysfunction. Two patients with identical BMI can have markedly different visceral fat areas and metabolic risks. In CRC surgery, visceral adiposity can also influence operative exposure, mesenteric thickness, technical complexity, and postoperative complications in ways that BMI does not fully capture. [5]

A single BMI measured at diagnosis is vulnerable to reverse causation. Occult CRC can cause anorexia, inflammation, reduced intake, and catabolism before diagnosis, lowering weight in patients who were previously overweight or obese. When these patients are classified only by diagnostic BMI, aggressive disease may be overrepresented in the normal or underweight categories, creating the appearance that overweight patients fare better. Weight history is therefore essential for interpreting observational associations. [6]

Sex, age, ethnicity, smoking, comorbidity, and treatment selection further complicate BMI-outcome associations. Women and men differ in fat distribution, older adults lose muscle despite stable weight, smokers often have lower BMI, and severe comorbid illness can influence both body weight and survival. Statistical adjustment reduces but does not eliminate these biases. The obesity paradox should consequently be treated as a hypothesis requiring careful causal analysis rather than as proof that excess adiposity is protective. [7]

The Obesity Paradox in Colorectal Cancer

Several clinical-trial and population-based studies have reported a nonlinear association between BMI and survival after CRC diagnosis. In stage III colon cancer trials, overweight patients have sometimes had outcomes similar to or better than those with normal BMI, whereas underweight and severe obesity more consistently identify higher-risk groups. Such findings may reflect nutritional reserve, lower prevalence of occult weight loss among overweight patients, treatment-selection effects, or unmeasured body composition. [8]

Postdiagnosis studies are equally heterogeneous. Prospective analyses and meta-analyses have suggested that being underweight carries a clear mortality disadvantage, while modest overweight is not consistently harmful. The interpretation changes when smoking, prediagnostic weight loss, and timing of weight assessment are considered. A patient who enters survivorship with BMI 27 after losing 15% of body weight is biologically different from a weight-stable patient with lifelong BMI 27. [9]

Stage-specific effects add further complexity. Prediagnostic BMI may relate differently to stage II, stage III, and metastatic disease because tumor burden, cachexia, treatment intensity, and competing mortality differ. Molecular tumor subtype may also modify associations between body size and CRC biology. Recent work linking BMI with molecular subtypes supports the idea that host phenotype and tumor biology interact rather than operate as independent prognostic domains. [10]

Visceral Adiposity

Visceral adipose tissue is metabolically active and drains partly into the portal circulation, exposing the liver to free fatty acids and inflammatory mediators. In CRC, visceral obesity may therefore influence both systemic metabolism and the immediate mesenteric tumor environment. CT-derived visceral fat area provides a direct estimate that is not confounded by muscle mass to the same degree as BMI. This makes it a more specific marker of central adiposity in research and potentially in clinical practice. [11]

Surgical studies show that visceral obesity can increase technical difficulty and may be associated with longer operations, conversion to open surgery, wound complications, or anastomotic risk. The magnitude of effect varies across cohorts, surgical



approach, tumor location, and definitions of obesity. Importantly, some patients with low or normal BMI can still have high visceral fat, while others with high BMI have predominantly subcutaneous adiposity. This discordance explains why BMI alone performs inconsistently as a surgical-risk marker. [12]

Visceral adiposity should not automatically be interpreted as a direct cause of poor oncologic survival. It can correlate with metabolic syndrome, diabetes, inflammation, and physical inactivity, which may mediate risk. Conversely, patients with advanced cachexia can lose visceral fat and appear metabolically lean despite aggressive cancer. Longitudinal imaging that tracks changes in visceral fat from diagnosis through treatment may therefore be more informative than a single baseline measurement. [13]

Skeletal Muscle, Sarcopenia, and Myosteatorsis

Sarcopenia originally described age-related loss of skeletal muscle but is now understood as a syndrome involving reduced muscle strength, quantity, or quality. In cancer studies, CT-defined low skeletal-muscle area is often used because routine imaging is available, although functional measures such as hand-grip strength and gait speed provide complementary information. Standardization remains challenging because sex-specific cutoffs, vertebral level, software, and population norms vary. [14]

Low skeletal-muscle mass is common in CRC and can occur across the BMI spectrum. Patients with obesity may have severe muscle depletion hidden beneath excess adipose tissue, creating sarcopenic obesity. This phenotype is clinically important because the patient carries both the metabolic burden of visceral fat and the limited physiologic reserve associated with muscle loss. Studies in CRC have linked sarcopenic obesity to recurrence, postoperative complications, and poorer survival. [15]

Muscle quality matters in addition to quantity. Myosteatorsis refers to fatty infiltration or reduced radiodensity of skeletal muscle on CT and is associated with impaired metabolic function and physical performance. Low muscle attenuation may identify patients with systemic inflammation, insulin resistance, or reduced reserve even when skeletal-muscle area is not severely reduced. Several CRC cohorts have reported adverse associations between myosteatorsis and outcomes. [16]

The biological relevance of muscle extends beyond physical strength. Skeletal muscle regulates glucose disposal, amino-acid metabolism, inflammatory signaling, and myokine production. During chemotherapy or postoperative stress, adequate muscle provides a protein reserve needed for immune function and tissue repair. Loss of muscle may therefore be both a marker of disease severity and a contributor to treatment intolerance. [17]

Sarcopenic Obesity

Sarcopenic obesity is not simply the coexistence of two diagnoses; it reflects an adverse body-composition phenotype in which high adiposity and low muscle interact. Obesity can promote inflammation and insulin resistance, while physical inactivity and cancer-related catabolism accelerate muscle loss. The resulting patient may have substantial body weight but limited functional reserve, making BMI particularly misleading. [18]

CRC studies using psoas or total skeletal-muscle measurements have associated sarcopenic obesity with shorter recurrence-free or overall survival in selected populations. A 2024 analysis found worse cancer-related relapse-free survival among patients with sarcopenia and visceral obesity, supporting the concept that muscle and fat should be evaluated together. Such findings require external validation but are clinically intuitive and potentially actionable. [19]

Cancer-Associated Weight Loss and Cachexia

Cancer cachexia is a multifactorial syndrome characterized by involuntary weight loss, muscle depletion, altered metabolism, and systemic inflammation that cannot be fully reversed by conventional nutritional support. Although it is most prominent in advanced disease, clinically relevant weight loss may begin before diagnosis in localized CRC. This matters because diagnostic BMI can become a surrogate for disease-induced catabolism rather than long-term adiposity. [20]

Weight-loss classification systems that combine percentage loss with current BMI have shown that the same absolute weight change has different prognostic implications depending on baseline size. A small patient losing 10% of body weight and a severely obese patient losing 10% may reach different absolute BMI values but both experience significant catabolic stress. Longitudinal weight data should therefore accompany BMI in outcome studies and routine oncology assessment. [21]

The prognostic value of weight loss also depends on timing. Prediagnostic loss may indicate tumor biology, postoperative loss may reflect surgical complications or reduced intake, and chemotherapy-associated loss may signal toxicity or progressive disease. In contrast, intentional fat loss with preserved muscle in a metabolically unhealthy survivor is a different process. Clinical



documentation should distinguish unintentional catabolism from planned weight management. [22]

Body Composition and Surgical Outcomes

Curative treatment for localized CRC relies heavily on high-quality surgery. Obesity can complicate exposure, mobilization, vessel ligation, and pelvic dissection, particularly in male patients with central adiposity and rectal cancer. Laparoscopic and robotic techniques remain feasible, but operative time, conversion, wound complications, and recovery may differ according to visceral fat and surgeon experience. Risk assessment based on imaging may be more informative than BMI alone. [23]

Anastomotic leak is a particularly important complication because it increases morbidity, prolongs hospitalization, delays adjuvant treatment, and may adversely affect oncologic outcomes. Meta-analytic data indicate that obesity can increase leak risk in colorectal surgery, although the effect varies by anastomotic site and study design. Visceral adiposity may better capture the technical and inflammatory factors contributing to this risk. [24]

Low muscle mass introduces a different surgical vulnerability. Sarcopenic patients may have reduced respiratory reserve, poorer mobility, impaired wound healing, and longer recovery. When sarcopenia and visceral obesity coexist, operative risk can reflect both technical difficulty and physiologic frailty. Preoperative CT therefore provides a unique opportunity to identify patients who may benefit from prehabilitation, nutritional optimization, and intensified postoperative support. [25]

Body Composition and Systemic Therapy

Chemotherapy dosing in adults is commonly based on body surface area. Historically, clinicians sometimes capped doses in obesity because of concern about toxicity. Current ASCO guidance recommends full weight-based cytotoxic dosing in obese adults when appropriate, including curative treatment, because empiric dose reduction can compromise efficacy and obese patients do not consistently experience greater toxicity when dosed according to actual body size. [26]

BMI, however, remains a poor measure of the metabolically active compartments into which drugs distribute. A patient with severe sarcopenia may receive a body-surface-area-based dose that is large relative to lean mass, potentially increasing exposure for drugs whose pharmacokinetics relate more closely to lean tissue. Evidence from oncology studies has linked low lean body mass to dose-limiting toxicity, although drug-specific relationships are complex and not yet sufficient to replace standard dosing rules. [27]

Oxaliplatin and fluoropyrimidine-based regimens remain central to adjuvant CRC therapy. Treatment benefit depends partly on maintaining adequate relative dose intensity, but toxicity can lead to delays, reductions, or early discontinuation. Body composition may help identify patients at higher risk for hematologic, gastrointestinal, or neurotoxic complications and could support earlier supportive-care interventions without automatically reducing initial dose. [28]

Targeted therapy and immunotherapy further weaken the assumption that body size alone determines treatment exposure. Many monoclonal antibodies and checkpoint inhibitors use fixed or weight-based regimens with wide therapeutic indices. Professional guidance recommends approved dosing rather than obesity-specific reductions. Future pharmacokinetic work may determine whether lean mass, visceral fat, albumin, or inflammatory state improves prediction for selected agents. [29]

Metabolic Health, Diabetes, and Inflammation

Obesity is heterogeneous. Some patients with high BMI have relatively preserved insulin sensitivity and low inflammatory burden, whereas others have diabetes, dyslipidemia, hypertension, fatty liver, and marked visceral adiposity. In CRC, this distinction may matter because hyperinsulinemia, inflammatory cytokines, and vascular disease can influence tumor biology, competing mortality, treatment tolerance, and postoperative recovery. Metabolic health should therefore be characterized directly rather than inferred from BMI. [30]

Diabetes has been associated with worse outcomes after CRC diagnosis in several cohorts and meta-analyses. Potential explanations include hyperinsulinemia, comorbidity, treatment modification, renal disease, infection risk, and competing cardiovascular mortality. The effect is not entirely independent of obesity, but diabetes provides clinically available information that can refine risk stratification and should be incorporated into body-composition research. [31]

Systemic inflammation also contributes to weight loss and muscle catabolism. C-reactive protein, albumin, and composite inflammatory scores have prognostic value in CRC and may interact with sarcopenia. A patient with low muscle and high inflammatory burden may represent active cancer cachexia, whereas a similarly sarcopenic patient without inflammation may have age-related or inactivity-associated muscle loss. These phenotypes may require different interventions. [32]

Physical Activity, Fitness, and Survivorship

Postdiagnosis physical activity has been associated with improved disease-free and overall survival in observational studies of



stage III colon cancer. Exercise may improve insulin sensitivity, reduce visceral fat, preserve muscle, lower cardiovascular risk, and enhance functional capacity. Although observational associations do not prove a direct antitumor effect, the breadth of health benefits makes structured activity an important part of survivorship care. [33]

Resistance exercise is especially relevant when muscle depletion is present. Aerobic training improves cardiorespiratory fitness and metabolic health, while progressive resistance training stimulates muscle protein synthesis and strength. Programs should be individualized around neuropathy, ostomy status, anemia, postoperative recovery, cardiopulmonary disease, and treatment schedule. The goal is preservation of function and metabolic health rather than indiscriminate weight loss. [34]

Cancer-survivorship guidelines encourage regular physical activity, healthy dietary patterns, and avoidance of excess body fat. For CRC survivors with obesity, weight management should protect lean mass and nutritional adequacy. Rapid caloric restriction during active treatment can be harmful if it worsens sarcopenia, whereas planned fat loss after recovery may improve cardiometabolic risk. Body-composition monitoring can help separate these scenarios. [35]

CT-Derived Body Composition as an Opportunistic Biomarker

Routine abdominal CT is ideally suited to opportunistic body-composition assessment because scans are already acquired for staging and surveillance. Skeletal muscle at the third lumbar vertebra correlates with whole-body muscle mass, while visceral and subcutaneous fat compartments can be segmented from the same image. Automated and semi-automated software is making this process faster and more reproducible. [36]

Clinical implementation still requires standardization. Different studies use different Hounsfield-unit thresholds, vertebral levels, sex-specific cutoffs, and definitions of sarcopenia or myosteatosis. Scanner calibration, contrast phase, slice thickness, and segmentation algorithm can also influence measurements. Before body composition becomes a routine prognostic report, consensus reference standards and external validation across ethnic and geographic populations are needed. [37]

Artificial intelligence may accelerate translation by automatically segmenting muscle and adipose compartments across entire CT datasets. Such algorithms can extract measurements at scale from retrospective trials and prospective care, enabling validation of thresholds and integration with electronic clinical data. The most useful models will likely combine imaging with age, sex, stage, treatment, laboratory variables, and longitudinal change rather than relying on a single area measurement. [38]

Toward Integrated Precision Risk Stratification

A practical CRC risk model could combine conventional variables with host phenotype. Tumor stage, histology, molecular markers, and treatment remain fundamental, but CT-derived muscle, visceral fat, recent weight change, diabetes, inflammatory markers, and functional status can add information about treatment resilience and competing mortality. Such a model would help distinguish biologically different patients who are currently grouped under the same BMI category. [39]

The host phenotype may also interact with tumor subtype. BMI has been associated with specific molecular features of CRC, and consensus molecular subtypes include a metabolically enriched CMS3 group and a stromal CMS4 group. These observations raise the possibility that metabolic exposures influence tumor behavior differently across genomic and transcriptomic contexts. Prospective studies should test interactions rather than simply adjust for molecular subtype as a confounder. [40]

Risk prediction should be dynamic. Body composition can change substantially during neoadjuvant therapy, surgery, adjuvant chemotherapy, recurrence, and survivorship. Loss of muscle during treatment may carry prognostic information independent of baseline status and may identify patients for early nutrition, exercise, symptom control, or treatment review. Longitudinal CT measurements are therefore potentially more informative than a single diagnostic snapshot. [41]

Dynamic models are also better suited to survivorship. A patient who regains fat but not muscle after treatment may develop sarcopenic obesity, while another may improve muscle and fitness despite stable BMI. Monitoring these trajectories aligns risk assessment with clinically modifiable targets and reduces reliance on weight alone. [42]

Prehabilitation and Supportive-Care Implications

Prehabilitation aims to improve physiologic reserve before major treatment through exercise, nutritional support, smoking cessation, and optimization of comorbid disease. CRC is particularly suitable because many patients undergo planned surgery and some receive neoadjuvant therapy that creates a defined preoperative interval. Patients with sarcopenia or poor functional status may derive the greatest benefit, although optimal program intensity and duration remain uncertain. [43]

Nutrition intervention should be phenotype specific. Patients with active weight loss and low muscle require adequate energy and protein, symptom management, and treatment of reversible causes of poor intake. Patients with stable obesity and preserved intake may need a different strategy focused on nutrient quality, metabolic control, and muscle-preserving activity. A universal



recommendation to lose weight during active cancer treatment ignores these distinctions. [44]

Multidisciplinary care is essential because body composition sits at the intersection of oncology, surgery, nutrition, rehabilitation, endocrinology, and geriatric medicine. Obese patients with CRC may also require attention to thrombosis, wound risk, obstructive sleep apnea, diabetes, and mobility. Dedicated pathways can address these issues without altering evidence-based oncologic treatment solely because of body size. [45]

Research Priorities

Future observational studies should collect prediagnostic weight history, serial weights, waist measures, CT body composition, physical function, diet, activity, and metabolic biomarkers. Without these data, investigators cannot reliably separate longstanding adiposity from cancer-associated wasting or determine whether visceral fat and muscle act independently. Standard definitions would also allow meaningful meta-analysis. [46]

Clinical trials should prospectively capture body composition and report treatment delivery, toxicity, dose intensity, recurrence, and survival by phenotype. Importantly, body-composition variables should be analyzed continuously as well as categorically because threshold-based definitions can lose information. Interaction analyses by sex, age, stage, and treatment may identify subgroups in whom host phenotype is most clinically relevant. [47]

Intervention trials should prioritize outcomes that matter to patients: postoperative complications, completion of adjuvant therapy, neuropathy burden, physical function, fatigue, hospitalization, recurrence, and quality of life. Changes in weight alone are insufficient. A program that produces modest weight change but preserves muscle and improves fitness may be more valuable than one that yields larger weight loss with muscle depletion. [48]

The field also needs implementation studies. Automated CT segmentation may be technically accurate but clinically irrelevant unless results are delivered in a form that changes care. Decision thresholds, referral pathways, and cost-effectiveness must be defined. The eventual goal is not to create another imaging number but to identify actionable phenotypes that trigger appropriate nutrition, exercise, metabolic, or treatment-support interventions. [49,50]

Anthropometric Alternatives and Functional Measures

Waist circumference and waist-to-hip ratio provide inexpensive estimates of central adiposity and often correlate more closely with visceral fat than BMI. Their use in oncology, however, is limited by postoperative anatomy, ascites, ostomies, measurement variability, and lack of standardized cancer-specific thresholds. They are best viewed as complementary bedside measures rather than replacements for CT-derived adiposity when imaging is already available. [2]

Functional measures add information that anatomy cannot provide. Hand-grip strength, chair-rise testing, walking speed, and cardiopulmonary fitness help distinguish a patient with low muscle quantity but preserved function from one with clinically important frailty. Current sarcopenia consensus frameworks prioritize muscle strength because it predicts adverse outcomes more directly than mass alone. Incorporating simple functional tests into CRC pathways could make imaging findings more actionable. [14]

Performance status remains essential but is relatively coarse and observer dependent. A patient may retain an Eastern Cooperative Oncology Group performance status of 0 or 1 despite early muscle loss, reduced aerobic capacity, or rapid weight decline. Combining performance status with objective body composition and strength measures could identify vulnerability before it becomes clinically obvious and before treatment toxicity forces dose modification. [18]

Rectal Cancer and Neoadjuvant Treatment

Rectal cancer deserves separate consideration because many patients receive months of neoadjuvant chemotherapy and radiotherapy before surgery. This interval can produce substantial changes in weight, muscle, and fat that are not captured by baseline BMI. Treatment-related diarrhea, pelvic symptoms, reduced activity, and systemic chemotherapy can accelerate muscle loss, while corticosteroids and inactivity may increase fat mass in other patients. Serial CT or MRI-based body-composition analysis could therefore identify deteriorating physiologic reserve before definitive surgery. [41]

Total neoadjuvant therapy has increased the duration and intensity of preoperative systemic treatment for locally advanced rectal cancer. Maintaining dose intensity and surgical fitness across this period is clinically important. Patients who develop progressive sarcopenia may require early nutrition, exercise, symptom control, or treatment-support interventions even when their total body weight appears stable. The concept of weight stability can be misleading when fat gain masks simultaneous muscle loss. [28]

Pelvic anatomy also magnifies the influence of central obesity. Visceral fat, a narrow male pelvis, and bulky mesorectal tissue may increase technical difficulty during total mesorectal excision. Robotic surgery can mitigate some ergonomic and



visualization challenges, but the host phenotype still affects operative planning and recovery. A combined model using pelvic anatomy, visceral fat, and muscle reserve could provide a more rational estimate of perioperative risk than BMI alone. [23]

Longitudinal Change as a Prognostic Signal

Static measurements are clinically convenient but biologically incomplete. Serial CT scans allow measurement of muscle and adipose change during treatment, and several studies suggest that ongoing muscle loss is associated with poorer outcomes even after baseline composition is considered. This dynamic information may capture active catabolism, inadequate intake, treatment toxicity, or progressive disease earlier than conventional weight monitoring. [50]

A useful longitudinal framework should distinguish four trajectories: stable muscle with stable fat, muscle gain with fat reduction, muscle loss with stable or increased fat, and combined loss of muscle and fat. These trajectories likely carry different implications for treatment tolerance and survivorship. The third pattern is especially important because BMI can remain unchanged while clinically relevant sarcopenia develops. [39]

Longitudinal assessment also creates an opportunity for intervention-response monitoring. If resistance training and nutrition support stabilize muscle attenuation or skeletal-muscle area, imaging can provide objective evidence of physiologic benefit. Such endpoints may be more sensitive than survival in early-phase supportive-care trials and can help establish whether a program changes the intended host phenotype. [43]

Special Populations and Clinical Interpretation

Older adults illustrate why BMI alone can be particularly misleading. Aging is accompanied by loss of muscle, redistribution of fat toward visceral depots, reduced anabolic responsiveness, and greater comorbidity. An older patient with a BMI in the overweight range may therefore have both sarcopenia and cardiometabolic disease. Comprehensive geriatric assessment, functional testing, and CT body composition can identify vulnerabilities that are invisible to weight-based classification. [14]

Sex-specific interpretation is also needed. Men and women differ in visceral fat accumulation, muscle mass, hormonal milieu, and the relation between BMI and metabolic risk. Uniform cutoffs can therefore misclassify risk, especially in populations that differ from the cohorts in which thresholds were developed. Validation should include sex-specific and ethnicity-sensitive reference values rather than assuming one universal definition of low muscle or high visceral fat. [37]

Patients with ostomies, chronic diarrhea, bowel obstruction, or treatment-related malabsorption require particular attention because weight and hydration can fluctuate rapidly. Apparent short-term weight change may reflect fluid balance rather than tissue loss. Repeated clinical assessment, dietetic review, and imaging-derived composition can help distinguish dehydration, fat loss, and muscle catabolism and prevent inappropriate dietary restriction in patients already at nutritional risk. [44]

Metastatic CRC presents a different balance of priorities from curative-stage disease. In advanced cancer, cachexia, symptom burden, corticosteroid exposure, and repeated treatment lines can dominate body-composition change. Prognostic information remains useful, but interventions should focus on preserving function, treatment tolerance, and quality of life rather than pursuing weight targets that may be unrealistic or harmful. [17]

Clinical reports should therefore avoid labeling patients simply as obese, normal weight, or underweight when more informative data are available. A concise phenotype such as high visceral adiposity with preserved muscle, sarcopenic obesity, or active weight loss with low muscle communicates more about operative risk and supportive-care needs. Standardized terminology could improve communication across oncology, surgery, radiology, nutrition, and rehabilitation teams. [45]

Conclusion

BMI remains useful for rapid population-level classification, but it is too crude to represent the biological diversity of patients with colorectal cancer. Visceral adiposity, skeletal-muscle mass, muscle quality, weight trajectory, metabolic health, inflammation, and physical function provide complementary information that can explain much of the apparent obesity paradox and reveal clinically important risk hidden within normal or elevated BMI. Routine CT offers a practical route to integrate these measures into oncology without additional imaging. The next generation of CRC prognostic models should combine tumor characteristics with dynamic host phenotyping and link each identified risk state to an actionable supportive or therapeutic strategy.

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