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Nota di contenuto	<p>Cover Page; Title Page; Copyright Page; Contents; I. Introduction; 1. Evolution of the fiscal balance in sub-Saharan Africa, current and past cycles; 1. Number of sub-Saharan African countries satisfying various macroeconomic performance and institutional quality criterial by decade; II. Literature Review; III. Empirical Strategy; A. Empirical Model and Identification; B. Data, Measurement, and Specification; IV. Results; A. Key Facts; 2. Cyclical properties of government spending, 1970-2008; 3. Robustness checks, system-GMM estimates and additional controls</p> <p>4. Cyclical properties of government spending by decade B. Factors; 5. Political factors, impact on procyclicality, 1970-2008; 6. Financing constraints, impact on procyclicality, 1970-2008; 7. Macroeconomic stability and fiscal space, impact on procyclicality, 1970-2008; 8. How can we explain the evolution of procyclicality over time in sub-Saharan Africa?; V. Conclusions and Policy Implications; Appendix; References; Footnotes</p>
Sommario/riassunto	<p>This paper documents cyclical patterns of government expenditures in sub-Saharan Africa since 1970 and explains variation between countries and over time. Controlling for endogeneity, it finds government expenditures to be slightly more procyclical in sub-Saharan Africa than in other developing countries and some evidence that procyclicality in Africa has declined in recent years after a period of sharp increase through the 1990s. Greater fiscal space, proxied by lower external debt, and better access to concessional financing, proxied by larger aid flows, seem to be important factors in diminishing procyclicality in the region. The role of institutions is less clear cut: changes in political institutions have no impact on procyclicality.</p>

2. Record Nr.	UNINA9910483140803321
Autore	de Graaf Jacqueline
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Nota di contenuto	Table of contents; Foreword; About the authors; List of abbreviations; 1. The Life History of ApoB Lipoprotein Particles; 1.1 Physiology of the ApoB Lipoprotein Particles; 1.1.1 Introduction; 1.1.2 Anatomy of the ApoB48 and ApoB100 Lipoprotein Particles; 1.1.3 The ApoB48 Lipoprotein Particles: Chylomicrons and Chylomicron Remnants Physiological Role; 1.1.4 The ApoB100 Lipoproteins Particles: VLDL, IDL, LDL; 1.1.5 Lipoprotein(a); 1.2 Heterogeneity in the ApoB Lipoprotein Particles; 1.2.1 Introduction; 1.2.2 VLDL Heterogeneity; 1.2.3 LDL Heterogeneity 1.2.4 The Atherogenic Lipoprotein Phenotype 1.3 The Fatty Acid Cycle and the ApoB Lipoprotein Particles; 1.3.1 Introduction; 1.3.2 FA Flux and the Adipocyte - the Role of ApoB48 and ApoB100 Lipoproteins; 1.3.3 Fatty Acid Flux in Hepatocytes; Summary; 1.4 The Hepatic Cholesterol Cycle and Regulation of ApoB Lipoprotein Particles; 1.4.1 Introduction; 1.4.2 Total Body Cholesterol Balance; 1.4.3 Regulation of Cholesterol Homeostasis in the Liver; 1.4.4 Regulation of LDL Particle Number in Plasma; 2. Diagnosis of the ApoB Dyslipoproteinemias: The ApoB Algorithm; 2.1 Introduction 2.2 The ApoB Diagnostic Algorithm HyperapoB: ApoB = 1.2 g/l versus NormoapoB: ApoB < 1.2 g/l; NormoapoB: NormoTG < 1.5 mmol/l versus HyperTG = 1.5 mmol/l; NormoTG NormoapoB; HyperTG NormoapoB: TG/ApoB = 10; HyperTG NormoapoB: TG/apoB = 10 and apoB = 0.75 g/l; HyperTG NormoapoB: TG/apoB = 10 and apoB < 0.75

g/l; HyperTG NormoapoB: TG/ApoB < 10; HyperTG NormoapoB: TG/ApoB < 10 and TC/ApoB = 6.2; HyperTG NormoapoB: TG/ApoB < 10 and TC/ApoB < 6.2; NormoTG HyperapoB: ApoB = 1.2 g/l and TG < 1.5 mmol/l; HyperTG HyperapoB: ApoB = 1.2 g/l and TG = 1.5 mmol/l

2.3 Limitations of any Diagnostic Algorithm3. The Primary ApoB Dyslipoproteinemias; 3.1 HyperTG NormoapoB; 3.1.1 HyperTG NormoapoB due to Increased Chylomicron Particles; 3.1.2 HyperTG NormoapoB due to Elevated Chylomicrons and VLDL Particles; 3.1.3 HyperTG NormoapoB due to Remnant Lipoprotein Disorder; 3.1.4 HyperTG NormoapoB due to Increased VLDL; 3.2 HyperTG HyperapoB due to Increased VLDL and LDL Particles; 3.2.1 Familial Combined Hyperlipidemia (FCH); 3.2.2 Sitosterolemia; 3.3.1 Severe Hypercholesterolemic Phenotype; 3.3.2 Treatment of the Severe Hypercholesterolemic Phenotypes
3.3.3 Polygenic Hypercholesterolemia3.3.4 CYP7A1 Deficiency; 3.3.5 Hypoalphalipoproteinemia; 3.3.6 Cholesteryl Ester Storage Disease (CESD); 3.3.7 Hypercholesterolemic NormoapoB Phenotype; 4. Secondary ApoB Dyslipoproteinemias; 4.1 Introduction; 4.2 Type 2 Diabetes Mellitus, Metabolic Syndrome, Abdominal Obesity; 4.2.1 HyperTG HyperapoB: Increased VLDL and LDL Lipoprotein Particles; 4.2.2 HyperTG NormoapoB: Increased VLDL Lipoprotein Particles; 4.2.3 HyperTG NormoapoB: Increased Chylomicron and VLDL Lipoprotein Particles
4.2.4 HyperTG NormoapoB: Increased Chylomicron and VLDL Remnant Lipoprotein particles

Sommario/riassunto

Now, based on the apoB algorithm, that is outlined and illustrated in this book, family physicians as well as cardiologists, endocrinologists and internists will be able to easily and accurately identify and treat these disorders. The apoB dyslipoproteinemias are major common causes of vascular disease. But until now, accurate diagnosis has not been possible. With just total cholesterol, triglycerides and apoB, all the apoB dyslipoproteinemias, with the exception of elevated Lp(a), can be identified using the apoB algorithm. The apoB app, which incorporates this algorithm, is available from both Apple and Android and is free. .
