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The Leptin-to-Adiponectin Ratio in Inflammation

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Abstract

Leptin and adiponectin are adipokines that have been shown to mediate the relationship between systemic inflammation and chronic diseases. However, the inconsistent reporting of leptin and adiponectin data by previous studies hinders the pooling of data across studies for cross-population comparisons or meta-analyses. As such, the aims of this brief are to (1) encourage researchers in the area of systemic inflammation to include standardized measures of leptin and adiponectin and the leptin to adiponectin ratio in future studies, and (2) recommend standardized reporting methods. Following standardized measurement and reporting methods will increase the likelihood that new data generated can be compared across studies and thus, knowledge about the impact of these adipokines on inflammation regulation can be advanced.

Keywords: Leptin; Adiponectin; Adipokines; Inflammation; Human studies

Abbreviations: CRP: C-Reactive Protein; LAR: Leptin-to-Adiponectin Ratio; TNF: Tumor Necrosis Factor; IL: Interleukin; ALR: Adiponectin-to-Leptin Ratio. EIA: Enzyme Immunometric Assay; ELISA: Enzyme Linked Immunoassay; IFMA: Immuno Flouro Metric Assay; QIA: Quantikine immunoassay; RIA: Radioimmunoassay

Introduction

Chronic systemic inflammation is associated with an increased risk for and a more rapid progression of multiple chronic diseases, including cardiovascular disease, diabetes mellitus, and kidney disease [1-6]. Identifying biomarkers and molecular contributors of chronic systemic inflammation early in its course may allow for earlier implementation of efficacious treatments, to prevent tissue and system damage, and thus reduce long-term disease ramifications [3]. Currently, the acute phase inflammatory biomarker c-reactive protein (CRP) is used in research and clinical practice [7-10] to help predict chronic disease and to guide treatment [11]. However, multiple factors, including certain medications, nutritional supplements, and acute injuries can cause CRP levels to be highly variable and unreliable [12].

Leptin and adiponectin are adipokines (hormones) made and released from adipose tissue that are involved in inflammation regulation. For example, leptin can activate various signal transduction cascades that result in the release of a host of other inflammatory mediators [13]. Leptin has been shown to have mostly pro-inflammatory actions, including up-regulating CRP, while adiponectin has been shown to have primarily anti-inflammatory actions, including down-regulating CRP [14-20]. Therefore, leptin and adiponectin are inflammatory biomarkers that may be more sensitive than CRP for detecting inflammatory status in clinical and research settings. Moreover, some studies have shown that the leptin-to-adiponectin ratio (LAR) has a higher diagnostic accuracy in detecting an inflammatory profile than leptin or adiponectin alone [21-24]. As such, the purpose of this brief report is to emphasize the importance of standardized assessment and reporting of leptin, adiponectin, and the LAR in future studies to facilitate more accurate comparisons across studies and data synthesis.

Background

Leptin and adiponectin are hormones made and released by adipocytes in adipose tissue that current literature describes as being mediators of inflammation and influencers of chronic diseases [25-29]. Leptin and adiponectin can be detected in tissue samples [30] however, they are most frequently measured in blood samples for clinical studies [29,31]. The proinflammatory actions of leptin are varied and include 1) inducing monocytes and macrophages to synthesize eicosanoids, 2) triggering of natural killer cell cytotoxicity, 3) stimulating chemotaxis in neutrophils, 4) activating proinflammatory cytokine production in B cells,

and 5) promoting Th1-cell immune responses (Th1 cells secrete tumor necrosis factor (TNF)-α, lymphotoxin, and interleukin (IL)-2) [19,32-35]. Conversely, the anti-inflammatory actions of adiponectin include inhibiting the production of proinflammatory cytokines, such as TNF-α and IL-6 by some immune cells (e.g., monocytes) [14,15,19,36,37]. Additionally, some studies report that adiponectin promotes the production of anti-inflammatory cytokines such as IL-10 [38,39].

The LAR has recently been identified as an important indicator of the collective actions of leptin and adiponectin and thus may be a more accurate biomarker of systemic inflammation than leptin or adiponectin alone [36,40,41]. While higher LARs have been shown to signify greater systemic inflammation and lower LARs have been shown to indicate less systemic inflammation [42,43], reference values have not yet been established.

Reporting Issues and Recommendations

There are several issues regarding the reporting of leptin and adiponectin in clinical trials. First, clinical studies assessing systemic inflammation often omit leptin and adiponectin as outcome measures or measures only one of these biomarkers [44-47]. A recent systematic review indicated that not only is the omission of these adipokines in clinical studies a concern, but in the majority of studies that quantified leptin and adiponectin, the LAR was not reported and therefore was not included in the analyses.[29] We recommend including leptin, adiponectin, and the LAR as outcome measures of inflammation in future studies focusing on the causes and treatments of chronic systemic inflammation.

Second, although leptin and adiponectin data, similar to other biomarker data, are often skewed and analyzed on a transformed scale, [48] a systematic review found that studies rarely included a description of the data transformation technique that was used [29]. Not knowing the data transformation technique used can make it difficult to compare results across studies and to pool data for meta-analyses. We recommend a standardized data transformation technique that involves a two-step process to normalize skewed data [49]. Transforming the variable into a percentile rank that results in a uniform distribution is step one. In the second step, step one results are modified using an inverse-normal transformation, guided by the mean and standard error of the original variable. This two-step process will transform any data set to a normal distribution with the same mean and standard error of the original data [49].

Third, there is variability in the unit of measurement of leptin and/or adiponectin reported by previous studies. Although the most frequently used measurement unit for leptin reported by previous studies is ng/ml, [24,50-53], mcg/l, [54] and pg/ml have been reported by other studies [55]. Similarly, the most commonly used measurement unit for adiponectin is mcg/ml, [24,45-47,50-54,56], however ng/ml, [57,58] mg/ml, [59,60] pg/ml, [55] and mg/L have also been used [44,61]. We recommend that the standardized measurement unit for leptin be ng/ml and the mcg/ml should be used for adiponectin. given that they have been the reporting units most frequently used by previous studies.

Fourth, some previous studies including leptin and adiponectin assessments have reported the adiponectin-to-leptin ratio (ALR), [62,63] while others have reported the LAR [41,42,64-66]. We recommend using the more commonly reported LAR versus the ALR. High levels of the ALR indicate less inflammation, while high levels of the LAR indicate more inflammation. Using LAR, instead of ALR, would allow for the natural association of values being indicative of a higher pro-inflammatory status. When the LAR is reported, it is often calculated by dividing leptin (ng/ml) by adiponectin (mcg/ml) [24,31]. We recommend continuing this practice in future studies.

Methods used for Measuring Leptin and Adiponectin

Information was taken from a systematic review by Rausch et al. (2020) with author approval [29]. Leptin and adiponectin have been measured using various methods. The 31 studies included in the systematic review reported the following methods for reporting leptin and adiponectin.[29] Several studies did not report the methods used for measuring leptin and adiponectin [29]. Moreover, it is unclear how much variation occurs between the different methods used.

Table 1: Methods used for measuring leptin and adiponectin.

Method Used	Number of studies reporting use (Obtained from Rausch et al., 2020)[29] (Obtained from Rausch et al., 2020)29	
	Leptin	Adiponectin
EIA	2	1
ELISA	5	15
IFMA	0	1
Time-Resolved IFMA	0	1
Luminometry	1	1
Luminex	1	0
QIA	0	1
RIA	4	5

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Fifth, complicating the ability to accurately compare levels of leptin and adiponectin across human studies is that the methods used to quantify these adipokines in previous studies have varied widely [29] Table 1. Given that it is still unclear if all methods for quantifying leptin and adiponectin yield similar results, additional studies are needed to make this determination.

Finally, various previous human studies have measured leptin and adiponectin in plasma/serum, saliva, hair, and/or urine [30,31,64-74]. There may be methods that would allow the comparison of levels of leptin and adiponectin across sample types, but additional studies are needed to explore this possibility. Using non-invasive methods to collect samples from sources other than blood to quantify leptin and adiponectin would allow researchers to analyze data from very young participants and compare them to other age groups. Moreover, parents may be more inclined to allow their children to participate in research studying inflammation across the lifespan if noninvasive methods for sample collection were used.

Conclusion

Taken together, to improve the accuracy of comparing leptin and adiponectin data across human studies, we recommend researchers investigating systemic inflammation consider including leptin, adiponectin, and the LAR as outcome variables in future studies, use a standardized transformation technique for skewed data (and report same), and report leptin and adiponectin data in standardized measurement units. Over the last 20+ years, information about how leptin and adiponectin influence inflammation regulation has increased. Yet, there is still much to be learned about how to use these important adipokines to guide disease diagnostics and treatment.

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Author Contributions

JR conceived the presented idea and wrote the manuscript. JM, and KH gave substantial feedback and assisted in manuscript writing and reviewing.

Conflict of Interest

The authors declare there are no conflicts of interest.

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