Success or Failure of Dental Implant Osseointegration: Is OsteoMacs polarization a key factor?

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BACKGROUND

Dental implant failure cannot always be explained by clinical risk factors. Recent literature suggests that immune cells are pivotal players in the integration of biomaterials and have a co-relationship within a set of osteal macrophages, known as OsteoMacs. These cells have been known to polarize quickly between a M1 pro-inflammatory and a M2 wound healing state during implant osseointegration. This plays a critical immune-surveillance role in the osteointegration of dental implant healing and bone homeostasis.

OBJECTIVE

The purpose of this study is to provide an overview of the current understanding of OsteoMacs and their role in early implant failure and osseointegration.

METHODS & MATERIAL

An electronic search was conducted from 2010 to 2020 using PubMed, Google Scholar and NYU electronic book library to identify relevant articles in English literature. Keywords were "osteomac", AND "implant" AND "failure". Four articles, 6 book chapters were selected.

RESULTS

M1 OsteoMacs release inflammatory cytokines while M2 OsteoMacs release wound healing cytokines. M2 OsteoMacs are associated with a higher peri-implant bone volume around stable implants while M1 OsteoMacs are implicated in foreign body rejection.
Biomaterials can increase M2 OsteoMac proportions through three mechanisms: (1) selective polarization of native OsteoMacs, (2) direct recruitment of native OsteoMacs with subsequent polarization and (3) direct recruitment of existing embryonic M2 OsteoMacs.
Certain biomaterial properties that favor M1 OsteoMacs include smooth, hydrophobic, hydroxylated nanoparticles and bio-inductive agents while rough, porous, hydrophilic, and hydrocarbon-based nanoparticles favor M2 OsteoMacs.
Vitamin D blocks pro-inflammatory cytokine release from M2 OsteoMacs and its deficiency is associated with early implant failure.

Figure 1

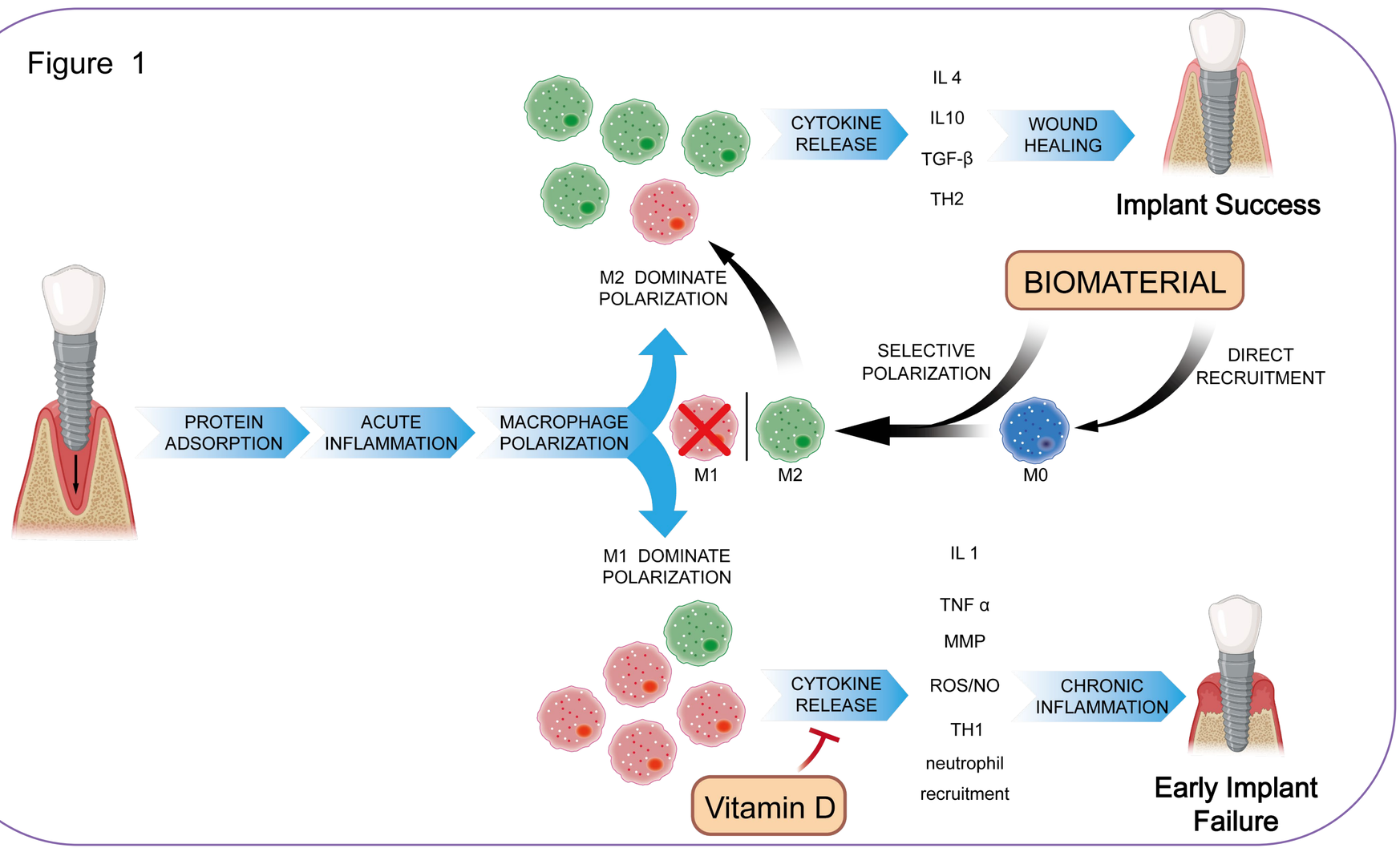


Figure 1b

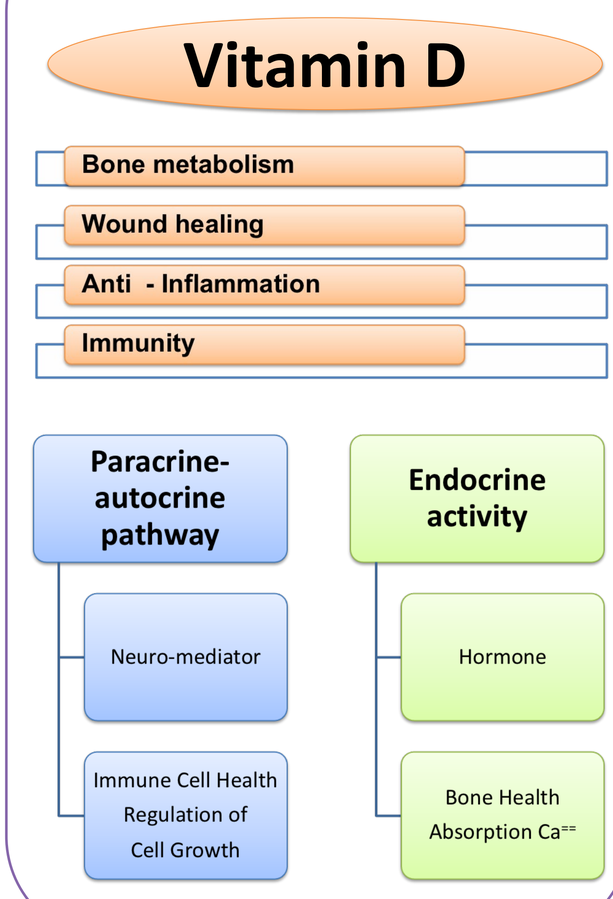


Figure 2

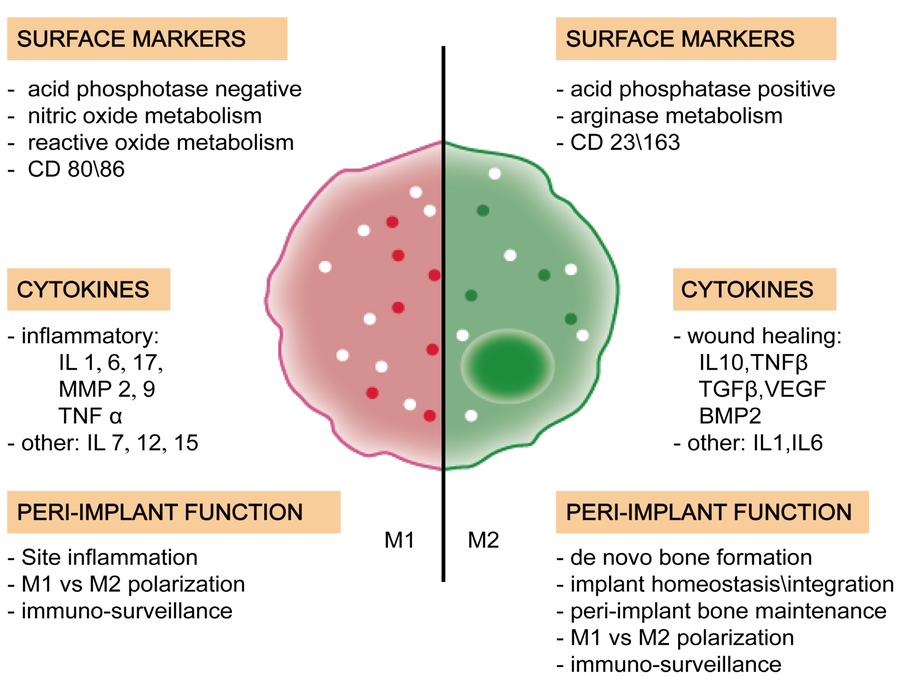
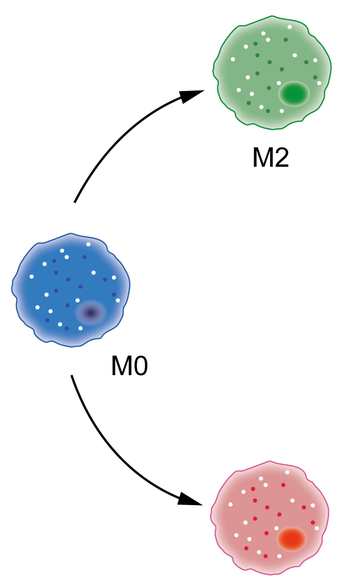


Figure 3



Disk Circular Rod Ellipsoid	Mesh Parallel Graded Grooved	Collagen Polyacrylic Acid	Carboxylic acid Fluorocarbon Nitro Ketone	Soft Substrate	Albumin VWF
Size	Geometry	Hydrophobicity	Surface Chemistry	Mechanics	Protein Adsorption
Ellipsoid Spherical Triangular Rod	Large Fibers Porous Fibers Grooved Rough	Hyaluronan Dextran Silicon Polystyrene	Sulfate Alkene Sulfone Amine Methy Group	Cyclic Stretch Static Stretch Stiff Substrate	IgG Fibronectin Fibrinogen Vitronectin

CONCLUSION

The ability of OsteoMacs to polarize between different states has been widely reported. Modulation of biomaterial surface properties and immune cell health to favor a desired OsteoMac state is a viable hypothesis that can explain the biology of early phases of successful implant osseointegration but also mechanisms of early implant failure.

REFERENCES

[1] Miron R.J, Bosshardt D.D. OsteoMacs: Key players around bone biomaterials. Biomaterials 2016 (82) 1-19.
[2] Insua A, Monje A, et al: Basis of bone metabolism around dental implants during osseointegration and peri-implant bone loss. J Biomed Mater Res Part A 2018 (105), 2075-2089.
[3] Jennissen H.P, A macrophage model of osseointegration. Curr. Dir. Biomed. Eng. 2016 (21), 53-56.
[4] Roszer T, The M2 Macrophage, Progress in inflammation research 86. Historical overview of macrophage polarization model. 2020 (86), 3-25.
[5] Roszer T, The M2 Macrophage, Progress in inflammation research 86. Mechanisms which control the size of M2 macrophage dominate tissue niches. 2020 (86), 99-111.
[6] Roszer T, The M2 Macrophage, Progress in inflammation research 86. Immune functions of the M2 macrophage: host defense, self tolerance, autoimmunity. 2020 (86), 115-132.
[7] Rayahin J.E., Gemeinhart R.A, Activation of macrophages in response to biomaterials. Macrophages, results and problems in cell differentiation. Chap 13, 317-351.
[8] Salehi, G, Behnamghader A, Mozafari M. Handbook of Biomaterials Biocompatibility. Cellular response to metal implants. Chap 20, 453-471.
[9] Mutreja I, Ye Z, Aparicio C. Handbook of Biomaterials Biocompatibility. Cell responses to titanium and titanium alloys. Chap 19, 423-452.
[10] Barbosa, J.N, Vasconcelos, D.P. Handbook of Biomaterials Biocompatibility. Macrophage response to biomaterials. Chap 3, 43-52.

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